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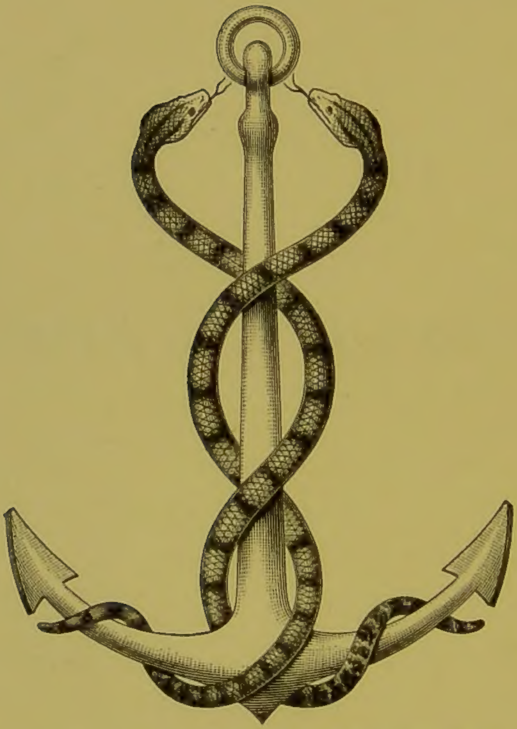
DISEASES OF THE STOMACH.



NUNQUAM ALIUD NATURA, ALIUD SAPIENTIA DICIT.

FUNCTIONAL & ORGANIC

DISEASES OF THE STOMACH.

BY

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PREFACE.

It is some years since a systematic treatise on diseases of the stomach was published in English. No apology is therefore necessary for the issue of this work. During the past few years great advances have been made not only in pathological chemistry, but in pathology generally; and these advances specially affect the study of functional and organic diseases of the stomach, inasmuch as the changes in the processes of digestion in disease have been studied during life, with the result of throwing great light not only on the symptoms, but also on the treatment of these diseases. Since it is possible to study changes in secretion, and, to a less extent, those in motor activity of the stomach during life, the functional disorders of the organ can be with advantage discussed in a separate treatise.

There can be but little doubt that the great difference between disorders of the stomach and those of the liver, pancreas, and salivary glands rests on the fact that the juices which these organs secrete are formed at some distance from the alimentary tract, so that the secreting structure of the glands is not affected by the direct irritant action of the food. It is this direct action of the food on the stomach, whereby both its secretion and its motor activity, as well as its absorption, are affected, which plays so great a part in the production of functional disorders of the organ, and accounts for the fact that functional disorders of the stomach are more commonly produced than those of the salivary glands or pancreas, or even of the liver. The fact of the stomach being the receptacle of the mixed and often uncooked food renders it liable to the invasion of micro-organisms, some of which decompose the stomach contents; while others, in certain conditions, invade the stomach walls.

The influence of chemical pathology on the study of disease cannot be over-estimated, and by its methods changes in secretion and metabolism are determined. As regards changes in secretion, the juices formed by the stomach and salivary glands are alone available for investigation during life. As regards metabolism, the study is a much more intricate one, inasmuch as changes in disease have to be determined from an investigation of the final products of the functional changes in the tissues, and in some cases at least it is not the final, but the intermediate, products which produce the symptoms of disease. Thus the metabolic changes of the liver in disease are in many respects a matter of conjecture, or at any rate are not so clear as the changes which occur in the stomach in disease.

In the present work I have adopted a method of discussing the diseases of the stomach which appears to me to be the most rational. Dealing first with the anatomy of the organ and the normal processes occurring in it, so far as our knowledge at present extends, the question of food is then considered from the point of view of a normal dietary and of the digestibility of food, and of the physiological effects of excess or diminution.

The pathology of indigestion of food is next considered, without taking into account whether it occurs in functional or organic disease; and the causes of indigestion to be ascribed to the food taken are discussed, together with the varying acidity of the stomach contents in disease, the gases generated, and the processes of digestion of the mixed food in the disordered stomach. One chapter has been devoted to the discussion of the methods of examination of the functions of the stomach in disease. This is a question that has been much neglected, in this country more particularly, but which is of very great importance both in the diagnosis and in the treatment of diseases of the stomach. Some of the methods which have been used are necessarily imperfect, and those given are mainly of clinical importance, and are as readily applied as the methods of examination of the urine in ordinary use.

I have considered the symptoms referable to indigestion of food in a separate chapter, without reference to the occurrence of organic disease, inasmuch as these symptoms are present both in functional and organic disease, and it is of importance to refer them, as far as is possible, to the changes which have occurred in the functions of the stomach.

Some exception may be taken to the classification which has been adopted in functional disorders of the stomach. In older works varieties of indigestion or dyspepsia have been discussed under the heading of "acid dyspepsia," "atonic dyspepsia," "flatulent dyspepsia," etc., a special class being made of neuroses of the stomach. Both from a pathological and from a practical point of view such a classification is of but little aid in the recognition and treatment of functional disorders of the stomach. Others, again, have made no classification, but have attempted to decide what functional disorder is present in each case. This would naturally be the most scientific method of dealing with functional disorders, if it were possible practically; but what is requisite when a large number of these cases have to be dealt with, often rapidly, and the object is to give relief and to promote cure, is such a classification as will enable one to readily distinguish the main features of a case. I have found it of great practical advantage, both in the recognition and treatment of functional disorders, to divide them into two classes, in one of which (gastric irritation) the main features are irritant symptoms of the stomach, produced by the food or food accessories which are taken; in the other of which (gastric insufficiency) the main feature is a primary deficiency in the functions of the stomach, leading to non-digestion of the food.

Under the heading of gastric irritation I have placed most of the forms of nervous dyspepsia or neuroses of the stomach, and considered them as gastric irritation with a prominence of nervous symptoms. For further discussion of these points, the text of the book must be consulted. The remainder of the work deals with organic diseases of the stomach and with treatment, which is considered separately. Owing to their importance, bleeding from the stomach and dilatation are treated of in separate chapters.

I am greatly indebted to Professor Thane, of University College, for much help and criticism in the anatomical account of the stomach, as well as for Figures 5 and 6, showing the structures touching the organ; also to Professor Halliburton, F.R.S., of King's College, for his kind help and criticism of the physiological and chemical part of the work; and to Dudley Cooper, Esq., M.R.C.S., for much help in preparing the statistics regarding ulcer and cancer, and in helping with the revision of the proofs.

The numerous works and original papers to which I am indebted are mentioned in the text.

SIDNEY MARTIN.

LONDON, 10 MANSFIELD STREET,
CAVENDISH SQUARE, *September* 1895.

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DISEASES OF THE STOMACH.

FUNCTIONAL AND ORGANIC DISEASES OF THE STOMACH.

CHAPTER I.

ANATOMY AND PHYSIOLOGY OF THE STOMACH.

Anatomy.

The stomach is a pear-shaped bag, with an opening (the cardiac) near its broader end into the œsophagus, and another opening (the pyloric) at its narrow end into the duodenum or first portion of the intestinal tract. It is one of the intra-peritoneal organs of the abdomen, and is capable under normal conditions of great changes in size and some changes in position. It is a specialised portion of the alimentary tract. It is the receptacle of the mostly undigested food, and produces a digestive secretion which differs from all other secretions in the body in possessing a free mineral acid, which acts as an antiseptic.

Form.—When quite empty, the form of the stomach is pear-shaped, and the anterior and posterior walls touch. When distended its form and divisions are more readily differentiated. The two chief portions are the cardiac on the left and the pyloric on the right; these are not separated in the human stomach by a line of demarcation, but the cardiac gradually passes into the pyloric region. There is an anterior and a posterior surface, and a lesser curvature, and a broader or greater curvature. The œsophagus enters the organ at the upper extremity of the lesser curvature, and to the left of the opening, the stomach is enlarged upwards, forming the fundus, which passes below into the greater curvature. The pyloric portion passes directly into the duodenum, the position of the pylorus being shown in the peritoneal coat by a sulcus, the pyloric sulcus, beneath which is the pyloric sphincter and the fold in the mucous membrane, called the pyloric valve. At a point in the greater curvature, a few inches from the pyloric sulcus, there is often a shallow groove, which marks the limit of the pyloric antrum; there may be a similar groove on the lesser curvature.

Size.—The size of the stomach varies according to the age and sex, and in the individual. It varies greatly according to the distention of the organ. At birth the fundus is absent. In the female the organ is, as a rule, smaller than in the male. According to Sappey, the following are the measurements of the stomach when moderately distended:—

In the greatest diameter, 24-26 cm. (9½-10½ inches).
From the lesser to the greater curve, 10-12 cm. (4-5 inches).
From front to back, 8-9 cm. (3-3½ inches).

When empty, the antero-posterior diameter disappears, and the other measurements are—

In the greatest diameter, 18-20 cm. (7-8 inches).
From the lesser to the greater curve, 7-8 cm. (2¾-3 inches).

These figures are averages: and in individual cases, variations will be found; the measurements being sometimes greater, sometimes less.

Capacity.—The capacity of the stomach varies. In some anatomical works, it is stated that the capacity is from 2½ to 4 litres (4 to 7 pints). This, however, is inaccurate. According to Ewald's measurements,[1] the capacity of the adult stomach may be as little as 250 cc. (9 ounces), or as much as 1680 cc. (59 ounces). The average is a little over a litre, or from 35 to 40 ounces; and if the capacity of the organ is 3 pints, the condition must be considered as pathological.

Position and relations (Figs. 1 and 2).—The stomach lies in the upper part of the abdominal cavity on the left side, extending from below the left vault of the diaphragm to the right side of the vertebral column. In the natural position it lies almost vertically, the œsophageal and pyloric openings being about 3 inches apart, and the anterior surface looking forwards and upwards, the posterior looking backwards and downwards; the lesser and greater curvature passing downwards and to the right. When fully distended (Fig. 1), the greater curvature comes forwards towards the anterior abdominal wall, so that the anterior surface looks more upwards and the posterior surface more downwards than in the undistended state.

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[1] "Klinik der Verdauungskrankheiten," Part ii. p. 86, 3rd ed.

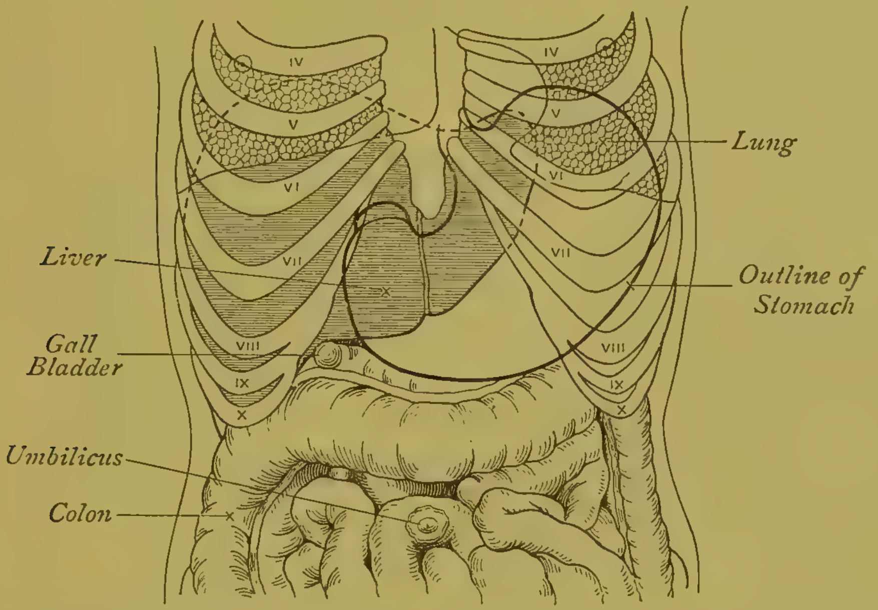

FIG. 1.—Position of the organs in the upper part of the abdomen. Front view. The highest points of the liver and fundus are somewhat too high in the figure. (After Luschka.)[1]

The stomach is held in position by the œsophagus as it passes through the diaphragm, by the fixation of the duodenum to the posterior abdominal wall, and by several peritoneal ligaments. The pylorus and duodenum are joined to the under surface of the liver by peritoneum, the gastro-hepatic and duodeno-hepatic parts of the lesser omentum, while the fundus is joined to the spleen by the gastro-splenic ligament, and the cardia is joined to the diaphragm by a small fold of peritoneum, the gastro-phrenic ligament. The result of this arrangement is that the stomach is suspended in the upper part of the abdominal cavity, and that there is no hindrance to the distention of the organs downwards, or upwards on the left side. The great omentum which is attached to the greater curve aids in keeping the organ in position, but does not limit its movements.

---

[1] H. von Luschka, "Die Lage der Bauch-Organe des Menschen," Carlsruhe, 1873, Plate I.

The *cardiac orifice* (cardia) is situated within one inch (2 to 3 cm.) below the œsophageal opening in the diaphragm, and on the left side of the body of the 10th dorsal vertebra (Fig. 2). It is about $4\frac{1}{2}$ inches (11 cm.) from the front wall of the trunk, and is in a line with a point one inch from the sternal insertion of the 7th left costal cartilage.

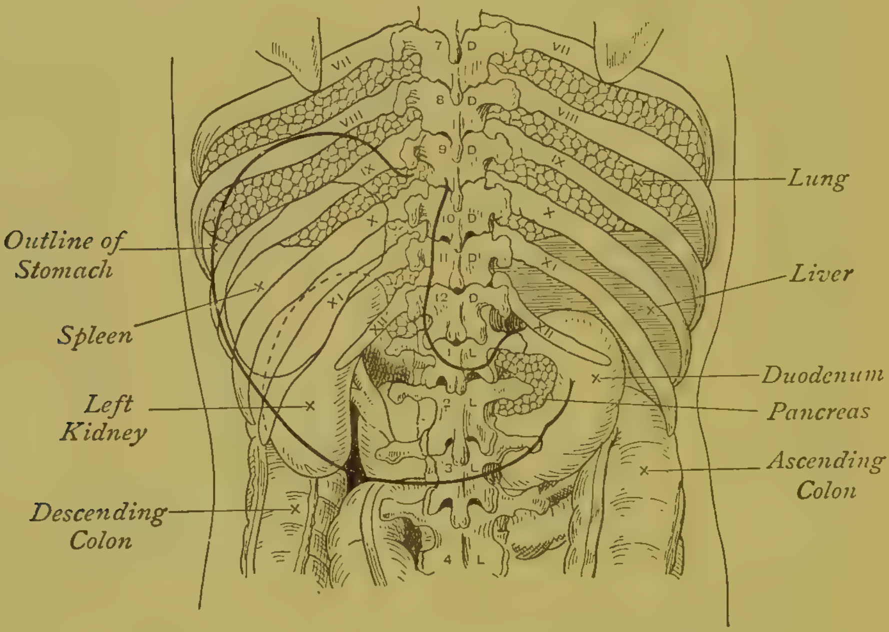

FIG. 2.—Position of the organs in the upper part of the abdomen. Back view. (After Luschka.)[1]

The *fundus* rises $1\frac{1}{4}$ to 2 inches (3 to 5 cm.) above the cardia, and is just below the lung. When greatly distended the fundus rests beneath the central tendon of the diaphragm and is just below the heart. In relation to the surface, the highest part of the fundus is opposite the 6th left chondrosternal articulation anteriorly (Fig. 1), and posteriorly it is opposite the 9th dorsal vertebra (Fig. 2).

---

[1] *Op. cit.* Plate II.

The *pylorus* is situated on the right side of the upper part of the 1st lumbar vertebra (Fig. 2), so that a horizontal section through the trunk at the level of the intervertebral disc separating the 12th dorsal and 1st lumbar vertebræ bisects the pylorus longitudinally (Fig. 3). On the anterior wall of the trunk, the pylorus is opposite a point somewhat below the xiphisternum and just outside the parasternal line (Fig. 1). In the undistended state of the stomach, the first part of the duodenum is horizontal and passes from left to right as well as backwards, but when the organ is distended the pyloric region moves forward carrying a portion of the duodenum with it: so that the first inch or so of the duodenum runs from front to back instead of right to left, and is seen cut transversely in a coronal section of the trunk (Fig. 4). During the normal movements of the stomach, this first inch or so of the duodenum is moving to and fro in the manner described. Like the stomach it is completely surrounded by peritoneum.

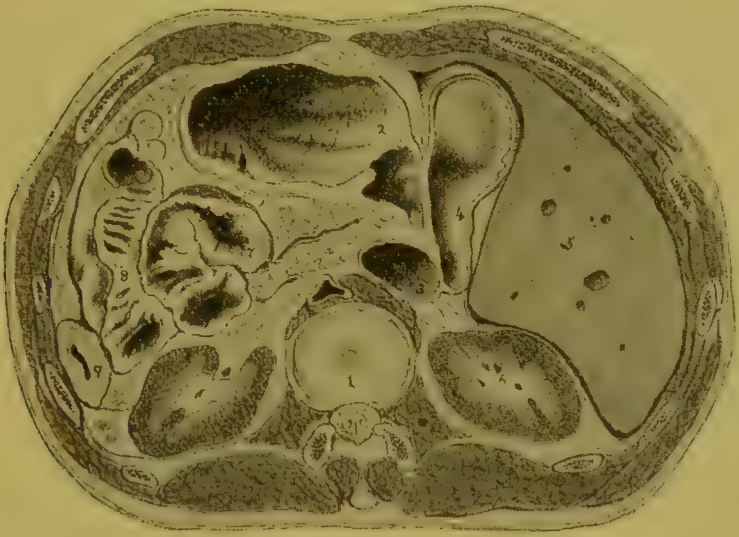

FIG. 3.—Transverse section of the trunk at the level of the disc between the 12th dorsal and 1st lumbar vertebræ. (From a model in the museum, University College.) 1, Intervertebral disc; 2, stomach near pylorus; 3, first part of the duodenum in transverse section; 4, gall-bladder; 5, liver; 6, right kidney; 7, small intestine; 8, colon; 9, colon.

The *lesser curvature* lies for the most part vertically. It begins at the cardia on the left side of the 10th dorsal vertebra, passes downwards on the left side of the bodies of the 11th and 12th dorsal vertebræ, and then crosses the body of the 1st lumbar vertebra, ending at the pylorus (Fig. 2). At the lower part it is crossed by a portion of the pancreas (Fig. 6).

The *greater curvature* varies in position according to the state of distention of the organ. When the stomach is distended the lower limit of the greater curve is marked by a transverse line drawn between the cartilages of the 9th ribs, or sometimes the 10th (Fig. 1). This line is two fingers' breadths above the umbilicus. When the stomach is empty, the lower line of the greater curve is much higher, being just observed below the edge of the liver; while the transverse colon occupies the position above indicated. Posteriorly, the greater curve of the distended stomach comes down as far as the 3rd lumbar vertebra (Fig. 2).

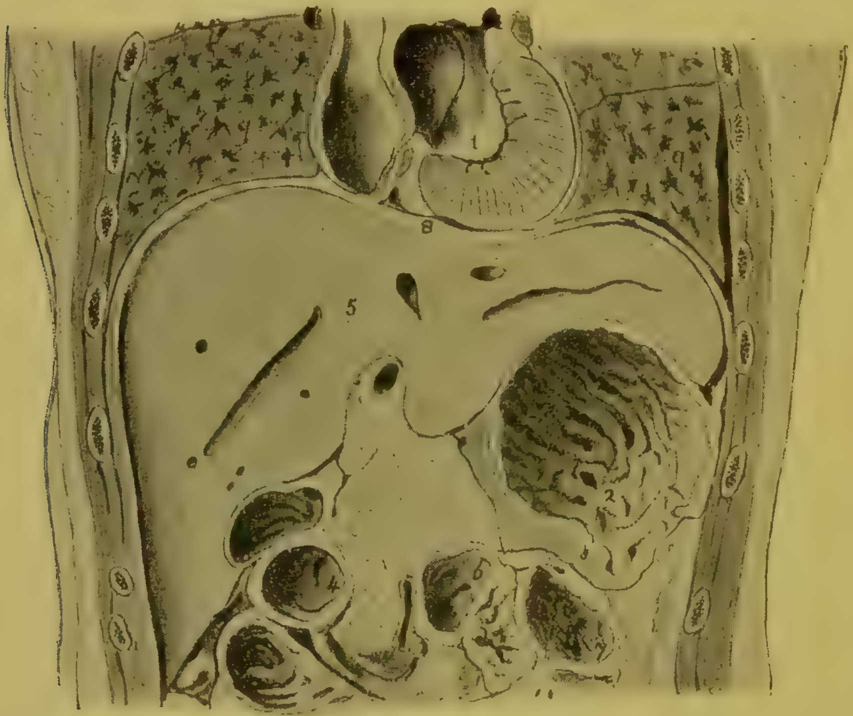

Fig. 4.—Coronal section of the trunk. (From a model in the museum, University College.) 1 heart; 2, stomach in transverse section; 3, gall-bladder; 4 and 6, duodenum; 5, liver; 7 colon; 8, diaphragm; 9, lungs.

The *anterior surface of the stomach* is in contact with the liver above, the anterior abdominal wall below and the diaphragm on the left (Fig. 5). The surface in contact with the liver is roughly oval, and extends from the cardia to the pylorus, which is situated just below the quadrate lobe. The surface in contact with the anterior abdominal wall, when the organ is distended, is triangular in shape (Figs. 1 and 5), the base of the triangle being opposite the 7th, 8th, and 9th rib cartilages. The surface in contact with the diaphragm extends upwards to the cardia and on to the posterior surface.

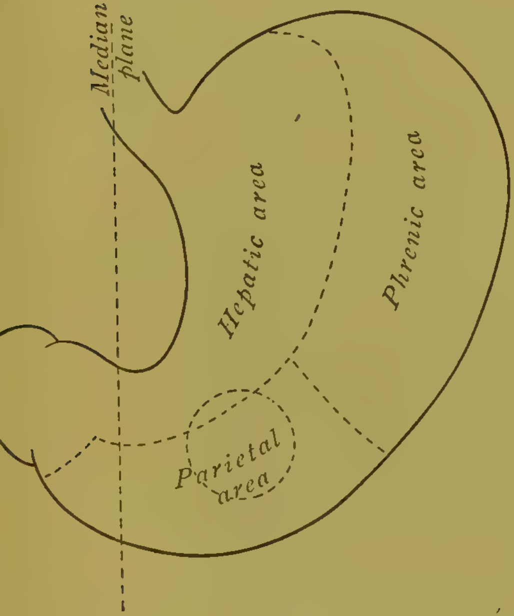

FIG. 5.—Diagram of the stomach in the natural position, showing the structures in contact with the anterior surface. The circle represents the position of the duodeno-jejunal flexure. (Thane.)

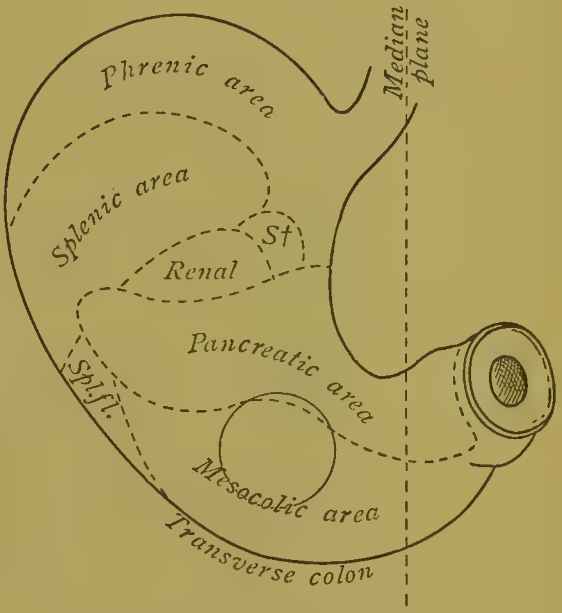

FIG. 6.—Diagram of the stomach in the natural position, showing the structures touching the posterior surface. St = suprarenal body. Spl.fl. = splenic flexure of the colon. The circle shows the position of the duodeno-jejunal flexure. (Thane.)

The *posterior surface of the fully distended stomach* lies against the following parts from left to right and from above downwards (Fig. 6): the diaphragm, whole internal surface of the spleen, all the suprarenal capsule, the upper part of the left kidney, and between the lower part of the kidney and the spleen, a portion of the colon (splenic flexure). The pancreas touches the posterior surface of the pylorus and crosses the lesser curvature in the direction of the spleen. The pancreas is not connected with the pylorus or with the commencement of the duodenum.

Structure.—The stomach is composed of four coats (Fig. 9): peritoneal (*serosa*), muscular (*muscularis*), submucous (*submucosa*), mucous (*mucosa*); the muscular and mucous coats forming the greater part of the wall of the organ. The outer *peritoneal coat* is a continuous covering for the organ, and beneath it lies a small quantity of connective tissue surrounding the vessels. The *muscular coat* is composed of unstriped muscle, and is divisible into three layers: a well developed longitudinal coat thickened along the greater and especially the lesser curves, and continuous above with the longitudinal coat of the gullet; a circular coat which is well developed all over the organ, and especially so at the pylorus, where it forms the pyloric sphincter, there being no special development of the circular coat at the cardia; and an internal slightly developed oblique coat mostly over the cardiac end of the organ. The *submucous coat* which lies just inside the muscularis consists of

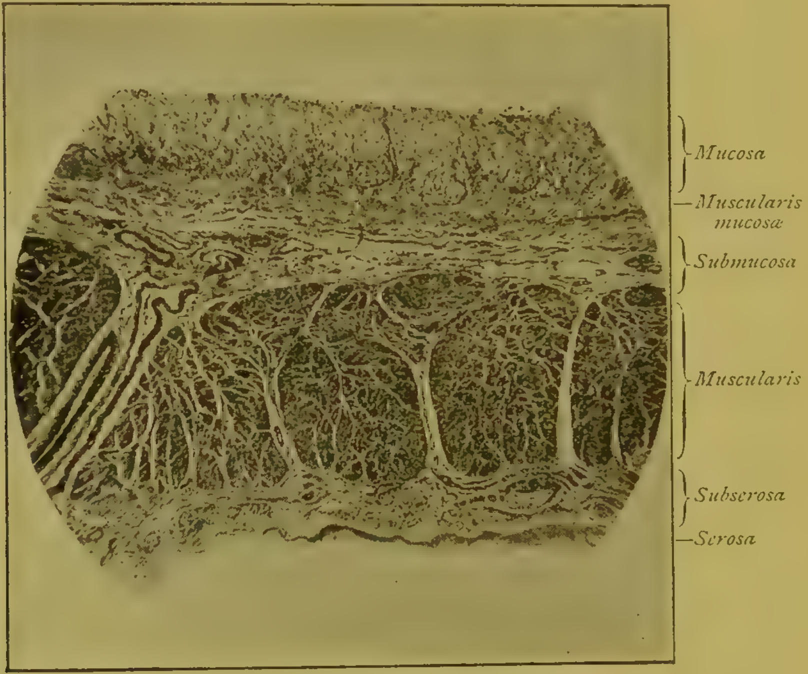

Fig. 7.—Section of the normal human stomach in the pyloric region. From a photograph, × 17. The specimen was obtained from an adult male. On the left of the figure an artery and vein are seen entering the submucosa.

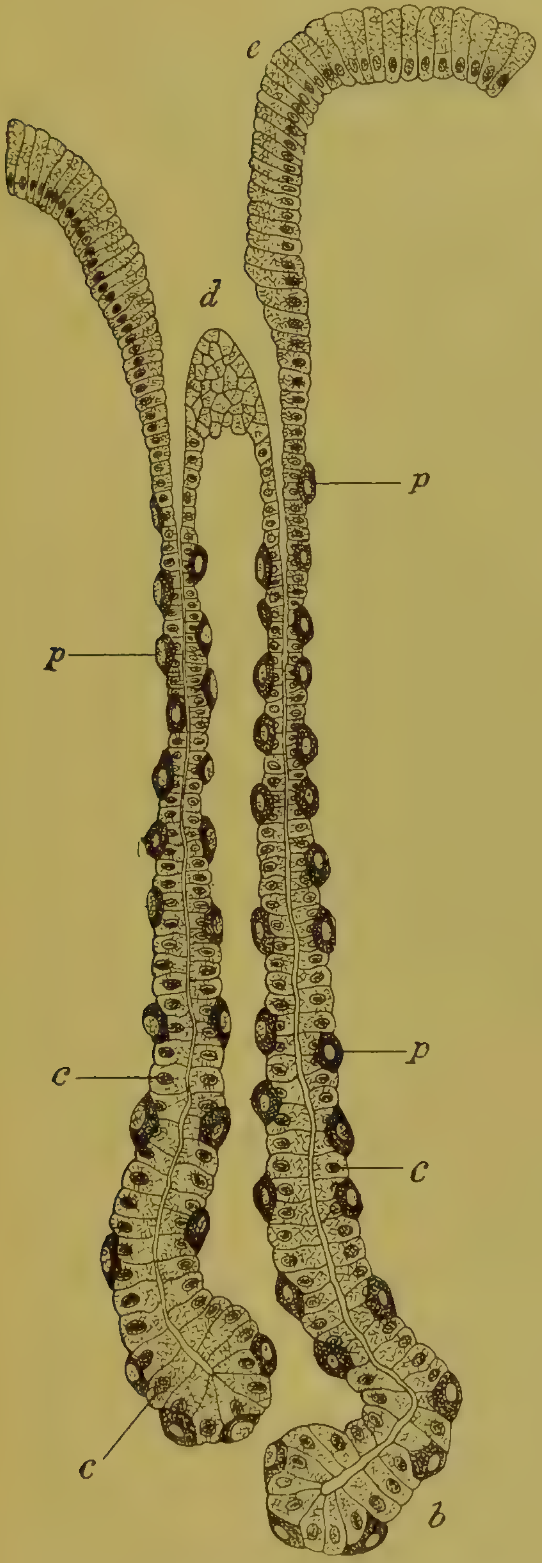

FIG. 8.—A cardiac gland from the dog's stomach (Klein and Noble Smith). *d*, mouth of the gland; *b*, fundus of one of the tubules: *e*, epithelium; *p*, parietal cells; *c*, central cells.

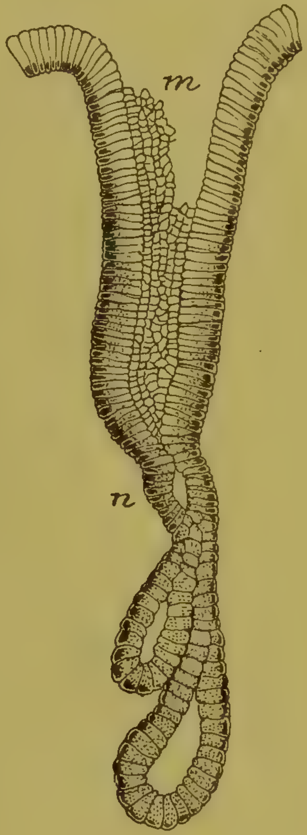

Fig. 9.—A pyloric gland from a section of the dog's stomach. *m*, mouth *n*, neck. (Ebstein.)

loose connective tissue, allowing great changes in extensibility of the inner mucous lining. In it the arteries break up to supply the mucous coat, and venous and lymphatic plexuses are formed by the vessels proceeding from the inner lining.

The *mucous coat* is separated from the submucous by a layer of muscular tissue, *muscularis mucosæ,* which is connected with the muscle fibres between the glands. The mucous membrane is thrown into longitudinal folds (rugæ), which are well marked, except at the pyloric end of the stomach. In some animals, *e.g.* the rabbit, the pyloric end of the stomach is sharply marked off from the cardiac, being thinner and more transparent; and in carnivora and in man the distinction between the two parts is evident to the naked eye. On the surface of the stomach numerous pits are visible under a magnifying glass, indicating the openings of the gastric glands. These are of two classes: the *cardiac* or *fundus,* and the *pyloric* glands. The cardiac glands are tubular, and several open on the surface by one duct. They are lined by two kinds of cells—one, forming the greatest number, are small, granular, nucleated cells, and secrete the pepsin (Hauptzellen of Heidenhain, chief, central or adelomorphous cells); the other, larger but also granular and nucleated, secrete the hydrochloric acid of the gastric juice, and are called parietal, delomorphous (Rollett), or oxyntic cells (Fig. 10), (Langley). The pyloric glands are also tubular and often branched at the extremity; they are lined by granular nuclear cells secreting pepsin or, more correctly, gastric juice without hydrochloric acid, the oxyntic cells being absent. The columnar cells which form the epithelial lining of the mucous coat of the stomach are in part goblet cells for the secretion of mucus. At the cardia there is a sharp demarcation between these cells and the stratified epithelium of the gullet, and at the pylorus the cells lining the duodenum are distinguished from those of the stomach by the presence of the hyaline border in the former.

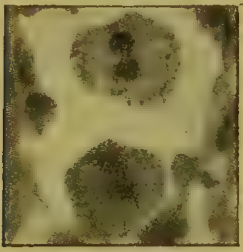
FIG. 10.—Oxyntic cells of the cardiac glands of the human stomach. From a photograph, × 350. The cells are not quite normal, as they are binucleated. A few chief cells are seen at the side of the alveolus. From a preparation hardened in osmic acid, and stained with logwood.

The stroma between the glands is composed of delicate connective tissue, with some adenoid tissue, supporting the blood vessels. Muscle fibres are also present (Fig. 11).

The submucosa contains collections of lymphoid tissue corresponding morphologically to those in the intestine, but they are not very conspicuous in the human stomach. They are observed chiefly in the pyloric region.

Vascular Supply.—The stomach is richly supplied with vessels (Fig. 12). The *coronary* branch of the cœliac axis passes from left to right along the lesser curve, supplying both anterior and posterior aspects of the organ, joining on the right branches of the *pyloric or right coronary artery* (a branch of the hepatic), and on the left anastomosing with the aortic œsophageal arteries. Along the greater curve there are two arteries which join—the *right gastro-epiploic*, which is the largest artery of the stomach, and is indirectly a branch of the hepatic; and the *left gastro-epiploic*, a branch of the splenic artery which also sends small branches to the cardiac end of the organ (*arteriæ gastricæ breves*). All the arteries of the

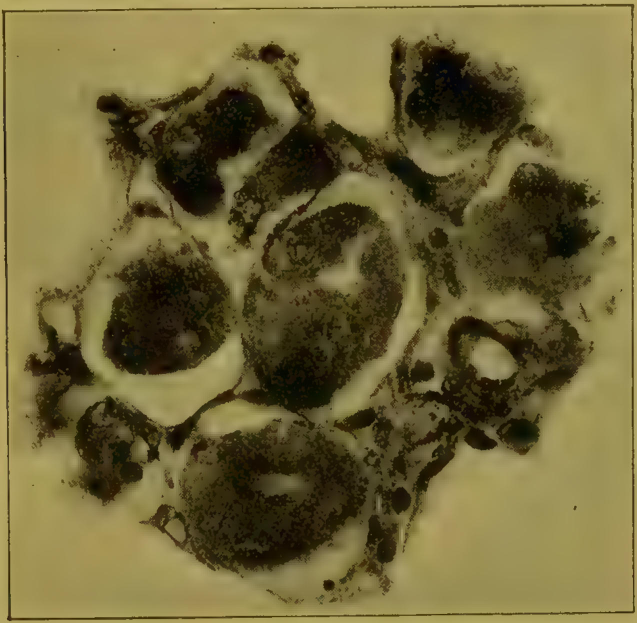

Fig. 11.—Transverse section of the glands of the cardiac region of a child's stomach. From a photograph, × 500. There is a very small amount of stroma, consisting chiefly of connective tissue cells with blood vessels. The alveolus in the centre of the figure shows one large (oxyntic) cell, and several small (chief) cells. From a preparation hardened in osmic acid and stained with logwood.

stomach freely anastomose. They enter the wall, and after supplying the muscular coats break up in the submucous coat and send numerous loops round the gastric glands in the mucosa.

The *veins* (Fig. 13) of the stomach all enter into the portal venous system, either directly into the portal vein, or into the splenic or mesenteric veins. They accompany their respective arteries, and bear the same names. The coronary vein is connected by branches with those of the lower part of the œsophagus, and is not infrequently a larger vein than the pyloric (Walsham).

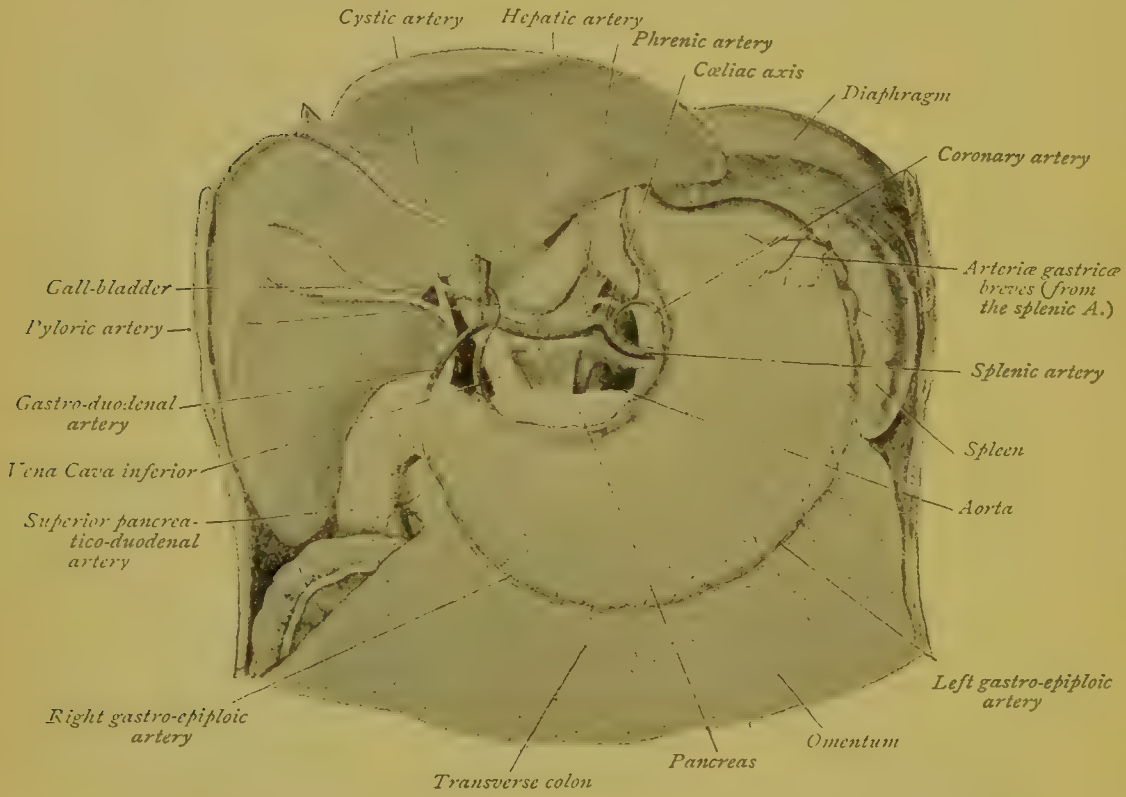

FIG. 12.—Arterial supply of the stomach. The liver has been turned upwards and the stomach pulled downwards somewhat. (Joessel.)[1]

The *lymphatics* of the stomach form two plexuses, one in the mucosa, the other in the submucosa. They then pass through the walls of the organ along the vessels, and go either directly or by means of the small gastric lymph glands which are situated along its curvatures to the retro-peritoneal glands and thence to the thoracic duct. Those accompanying the right gastro-epiploic vessels, enter the lymph glands at the head of the pancreas; those accompanying the left gastro-epiploic vessels enter glands in the hilus of the spleen.

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[1] G. Joessel, "Lehrbuch der topograph.-chirurg. Anatomie," ii. Th., 2 Abth.; "Der Bauch," Fig. 64, p. 266. Bonn, 1892.

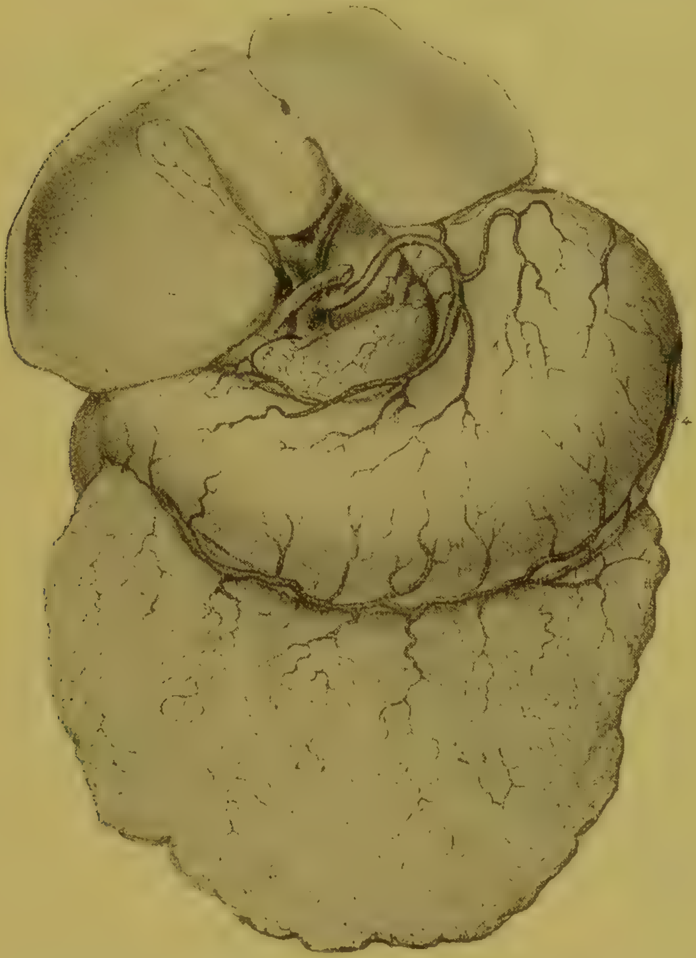

FIG. 13.—Venous supply of the stomach. 1, coronary vein; 2, pyloric vein; 3, right gastro-epiploic vein; 4, left gastro-epiploic vein; 5, portal vein. (Walsham.)[1]

The blood supply of the stomach comes therefore from the aorta by means of the cœliac axis, and the venous blood passes to the portal vein going through the liver, while the lymphatics go through glands to the thoracic duct. The passage of the blood through the liver and of the lymph to the thoracic duct is connected with the absorption processes which occur in the stomach.

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[1] Walsham, "Observations on the Coronary Veins of the Stomach," *Journ. Anat. and Physiol.*, London, vol. xiv. p. 399.

Nervous Supply of the Stomach.—The stomach is supplied by the vagus and the sympathetic nerves. The right vagus branches out on the posterior aspect, and the left, on the anterior; while branches of the sympathetic are supplied by the splanchnics and the cœliac plexus. The left vagus also supplies branches to the liver. Only about one-third of the trunk of the right vagus ends in the stomach, supplying the left half of the posterior surface, while the greater portion of the nerve passes to the semilunar ganglion. The right half of the posterior surface is supplied mainly by the sympathetic. Besides this there is in the muscular coat the plexus of Auerbach, containing ganglionic cells, and in the submucous coat the similar plexus of Meissner, both of which no doubt play some part in the physiological processes taking place in the organ.

PHYSIOLOGY.

Functions of the Stomach.—The physiological processes taking place in the stomach are the digestion of the proteid and albuminoid substances by means of the gastric juice; the mechanical and chemical breaking up of the masticated food; its propulsion into the small intestine; and the absorption of certain substances. The processes are therefore divisible into three classes:

1. Chemical.
2. Mechanical.
3. Absorptive.

The normal processes depend chiefly on the nervous mechanism of the stomach which governs the secretion of gastric juice and the movements of the organ, and by means of which the vascular supply is regulated.

Nervous Mechanism of the Stomach.—The stomach is directly connected with the central nervous system by the vagus nerves, and is not only supplied by the branches of the sympathetic from the solar plexus, but it has a ganglionic system of its own in Meissner's plexus in the submucous coat and in Auerbach's plexus in the muscular coat. The exact part the nerves play in the conduction of impulses to and from the stomach is not known; nor have the exact functions of the local ganglionic centres been discovered; but some ascertained facts are not without import.

Division of the vagus nerves (thus severing the most important connection of the stomach with the central nervous system) causes a momentary contraction of the pyloric sphincter and of the cardiac orifice, but does not completely remove the normal movements of the organ, although it diminishes them. Such an operation, moreover, although it does not inhibit the secretion of gastric juice, causes a pallor of the mucous membrane during digestion, instead of the increased vascularity which normally occurs. These results mean that the stomach is not wholly dependent on the higher part of the central nervous system for its movements or for the secretion of gastric juice. Stimulation of the end of the vagus nerve connected with the stomach produces after a time some movement; but not a very well marked one; while such a stimulation produces no secretion of gastric juice. This last result is in direct opposition to the case of the submaxillary salivary gland, for stimulation of the chorda tympani nerve produces a copious secretion of saliva. No effect on the movements or on the secretion of gastric juice has been observed by section or stimulation of the sympathetic nerves supplying the stomach. It is therefore evident that the mechanical movements of the stomach and the secretion of gastric juice are, to a great extent, independent of the central nervous system, and are possibly dependent on a local nervous mechanism. It can only be conjectured that the local nervous mechanism is the plexus of Meissner or of Auerbach or both, but this is very doubtful. When removed from the body of a recently killed animal and placed in an appropriate warm chamber, the stomach shows automatic movements. These are regular and commence at the greater curve near the cardia, progressing towards the pylorus.[1]

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[1] Hofmeister and Schütz, *Arch. f. exper. Path. u. Pharmakol.*, Leipzig, 1886, Bd. xx. p. 11.

It was mentioned above that section of the vagi caused pallor of the mucous membrane during digestion instead of the usual rosy colour; this appears to show that the vascularity of the organ during digestion is a reflex effect, the vagus being the afferent nerve, and the sympathetic the efferent. At any rate it must be granted that the increased vascularity of the organ during digestion is not solely due to the presence of food or of its chemical elements, but to the action of a nervous mechanism, since it is a prolonged effect, and is necessary not only for the absorption of certain food products, but for the manufacture of the gastric juice and for a continuance of the automatic movements. But little is known of the influence of the nervous system on the secretion of gastric juice. Stimulation of the vagus does not produce secretion: section of both vagi, of the splanchnic nerves, and extirpation of the cœliac ganglion does not abolish secretion. From a practical point of view it is, however, noteworthy that gastric juice is secreted in response to many stimuli applied to the mucous membrane. Such an inert body as a pebble will cause a flow of gastric juice, and electrical stimulation of the mucous membrane will have a similar effect. Of chemical stimulants, swallowed saliva, dilute alkalies, sodium chloride, as well as food, cause a flow of secretion; facts which are of prime importance in dietetics. The secretion of gastric juice is also effected by reflex action. This was very evident in a case observed by Professor Richet of Paris. In a woman who had gastrostomy performed for an impermeable stricture of the œsophagus (caused by a corrosive) it was noticed that a flow of gastric juice followed the introduction into the mouth of any substance causing a flow of saliva. There is no doubt a close nervous connection between the individual glands which secrete digestive juices and between these glands and the special senses of sight, smell, and taste, which are intimately connected with the taking and enjoying of food. It is certain that there is no secretory nerve directly supplied to the stomach glands from the central nervous system, although there is some evidence of a nervous connection between the central nervous system and the secretion of gastric juice. Physiological research tends to show that all secretion in highly specialised animals is more or less under the control of the nervous system: in the stomach the nervous mechanism is probably mainly a local one situated in the walls of the stomach and brought into action in response to mechanical, chemical, and other stimuli applied to the mucous membrane. The same must be said of the muscular contractions of the stomach: the modified peristalsis of the stomach, although more directly connected with the central nervous system than the secretion of gastric juice (since stimulation of the vagi affects the contractions of the stomach), yet goes on in a full stomach after section of both vagus nerves. The local nervous mechanism of the muscular contractions of the stomach possibly exists in Auerbach's plexus and ganglia situated in the muscular coat.

Vomiting is a reflex effect produced by many different stimuli. The actual act of vomiting is accompanied, first, by a flow of saliva and a pallor of the skin, with or without increased diaphoresis; secondly, by a deep drawn inspiration associated with a spasmodic contraction of the diaphragm, followed by closure of the glottis; thirdly, by a sudden contraction of the abdominal muscles accompanied by the opening of the cardiac orifice and some contraction of the stomach itself.

Vomiting is dependent on the integrity of a nervous centre in the medulla oblongata. This centre may be directly affected either by disease (cerebral) or by drugs, and this direct stimulation may produce an ejection of the stomach contents. The centre is, however, usually affected reflexly.

In the process of vomiting described above, the different stages are the result of the effect of the stimulus on the vomiting centre, which discharges along the secretory nerves of the salivary glands, causing a flow of saliva; which affects the neighbouring respiratory centre producing the deep inspiration, the closure of the glottis, the spasmodic contracture of the diaphragm, and the energetic expiratory contraction of the abdominal muscles.

The parts of the body from which vomiting is excited reflexly are as follows:—from the fauces, soft palate, and pharynx through the glosso-pharyngeal nerve; from the stomach, lungs, liver, and gall-bladder by means of the vagus nerves; from the uterus, kidneys, peritoneum, and intestine by means of the sympathetic fibres (visceral nerves) supplied to them. Vomiting is also excited by great pain, the reflex afferent impulse passing to the vomiting centre by an ordinary sensory nerve; and it is perhaps partly in this way that the passage of a gallstone or a renal calculus, or peritonitis and hernia produce vomiting. In some cases of disease it is not always easy to say what particular pathological factor produces the vomiting, as, besides local disease of an organ, there may be an inflammatory disease, the chemical products of which may produce emesis by acting directly on the centre in the medulla.

To sum up this part of the subject, it may be said that the central nervous system has no direct influence on the secretion of gastric juice or on the mechanical movements of the stomach; both these processes are largely automatic, and are probably dependent on the ganglia present in the walls of the stomach. The central nervous system is, however, connected with both these processes, since the secretion of gastric juice may be effected reflexly by the mere flow of saliva, by emotional disturbance, and by excitation of the special senses. The automatic movements are in closer connection with the central nervous system than the secretion of gastric juice.

The physiological processes in the stomach—*mechanical*, *chemical*, and *absorptive*—will now be considered seriatim.

Mechanical Processes in the Stomach; Movements.—In the intervals of digestion the normal stomach, being empty, is contracted, its mucous membrane lying in folds or rugæ and its muscular coat being in a state of more or less tonic contraction. Soon after the entrance of food, the movements begin and continue during the whole time of digestion; at first slight, they gradually increase in intensity. The first muscular action of the stomach is the opening of the cardiac orifice to allow the entrance of the food. This occurs by a contraction of the longitudinal muscular fibres surrounding the orifice. It is a reflex act, the proper performance of which depends on the integrity of the vagus nerve. Section of the vagus causes a spasmodic contraction of the cardiac orifice, the swallowed food collecting in the lower part of the œsophagus, and not entering the stomach. Another similar action is the opening of the pylorus dependent on the relaxation of the circular muscular fibres forming the sphincter; this relaxation appears to occur at irregular intervals to allow of the exit of the liquid food, and it occurs the more frequently the more liquid the condition of the food is. Its nervous mechanism is not exactly known. Section of the vagi causes a spasmodic contraction of the pyloric sphincter. The general movements of the stomach are of two kinds—the *circular* and the *churning*. The circular movement, due chiefly to the contraction of the circular muscular coat, is most powerful at the pyloric end; it is similar to the peristaltic wave in the intestine. It is the movement which occurs when the stomach is stimulated electrically, although in this case it is only local. By the circular movement the food is passed from one end of the organ to the other. The *churning* movement takes place near the mucous membrane, and by its means the particles of food near the membrane are driven towards the centre of the stomach. The effect on the food of these movements is to make it pass from the cardia to the pylorus and then back again, while it is also passing from the mucous membrane towards the centre of the organ (p. 29).

The stomach during digestion is practically a closed bag suspended between two more or less fixed points, viz. where the œsophagus passes through the diaphragm and at the duodenum. This fixation of the two ends aids the movements of the organ. By these movements the masticated food is mixed, the mixing becoming more complete as the proteid constituents of the mixed food are dissolved. At certain intervals the pylorus opens and part of the liquid contents of the stomach are ejected into the duodenum. But where the food taken is chiefly solid, most of it remains in the stomach until digestion is completed, when it is propelled into the duodenum *en masse*.

The movements of the stomach are diminished by violent exercise after a meal and cease during sleep (Busch). Violent emotions, it is said, will also cause them to cease for a time; this, however, is only a matter of conjecture from the fact that violent emotions will inhibit the digestion of food in the stomach, but this might occur also from an inhibition of the secretion of gastric juice as well as of the muscular movements of the organ.

Chemical Physiological Processes in the Stomach.—The *secretion of the gastric juice* takes place both in the glands of the cardiac and in those of the pyloric region; the former producing gastric juice, containing both pepsin and hydrochloric acid; the latter, gastric juice containing pepsin but no hydrochloric acid. The acid is formed by the large parietal or oxyntic cells previously described, which swell up during secretory activity of the glands; the pepsin is formed in the chief cells, which become very granular in the intervals between secretion, and discharge their granules during secretion (Langley). The hydrochloric acid[1] is secreted by the cells as such, and must of necessity be formed from a chloride and presumably from sodium chloride. It was suggested by Maly[2] that lactic acid is the means by which this salt is decomposed, setting free the acid, according to the following equation:—

$$\underbrace{2\,NaCl}_{\text{Sodium chloride.}} + \underbrace{2\,C_3H_6O_3}_{\text{Lactic acid.}} = \underbrace{NaCl}_{\text{Sodium chloride.}} + \underbrace{NaC_3H_5O_3}_{\text{Sodium lactate.}} + \underbrace{C_3H_6O_3}_{\text{Lactic acid.}} + \underbrace{HCl}_{\text{Hydrochloric acid.}}.$$

Although this might occur when lactic acid is present in the stomach contents, yet such a transformation cannot be considered the normal method of secretion by the parietal cells. It is more probable (Maly) that di-sodium hydrogen phosphate is decomposed by means of carbonic acid (CO_2), sodium hydrogen carbonate and sodium di-hydrogen phosphate being formed, according to the following equation:—

$$Na_2HPO_4 + CO_2 + H_2O = NaHCO_3 + NaH_2PO_4.$$

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[1] At first it may appear an anomaly that a free mineral acid should be secreted by an animal cell. The secretion of hydrochloric acid by the gastric glands is not the only phenomenon of the kind in nature. A gasteropod (Dolium galea) secretes free sulphuric acid in its "saliva" (quoted by Halliburton, "Chemical Physiology," p. 60; see also Bunge, "Lehrbuch der physiolog. und patholog. Chemie," 3rd ed., 1894, p. 144). The pupa of the Puss moth (Dicranoura vinula) secretes potassium hydrate (KHO) to enable it to emerge from its covering (Oswald Latter, *Trans. Entom. Soc. London*, 1892, part iv. (Dec.) p. 287).

[2] *See* Gamgee, "Text-Book of Physiological Chemistry," vol. ii. p. 111.

And that the sodium di-hydrogen phosphate reacts on sodium chloride forming free hydrochloric acid, according to the equation—

$$NaH_2PO_4 + NaCl = Na_2HPO_4 + HCl.$$

The glands are acid only in their upper parts; in the deeper, they are alkaline.

A very important point regarding the secretion of pepsin, and one that probably has some importance in cases of disease of the stomach, is that the ferment is not secreted as such, but is preceded by a mother-ferment, pepsinogen, which exists in the cell in the form of the granules previously described. This pepsinogen has no ferment action, and is readily transformed into pepsin by the action of dilute hydrochloric acid and of warmth. Both pepsin and pepsinogen are destroyed at a temperature of about 55° C. (Langley).

Composition of the Gastric Juice.—The gastric juice contains two ferments (pepsin and a milk-curdling one), dissolved in water containing mineral salts and free hydrochloric acid. A small amount of mucus is also present. The amount of hydrochloric acid varies from 0·02 per cent to 0·2 per cent; the amount of pepsin is also very variable, as shown by the activity of different specimens of juice. The amount of salts, consisting of chlorides of sodium, potassium, and calcium, and of phosphates of calcium, magnesium, and iron, is about 0·2 per cent.

The *properties of the gastric juice* are three in number:—

1. It possesses a milk-curdling ferment (rennin).
2. It possesses a proteolytic ferment (pepsin) which acts in conjunction with free hydrochloric acid.
3. Owing to the presence of a free mineral acid, it possesses antiseptic qualities (p. 70).

Chemical Processes of Digestion in the Stomach.—The *rennet or milk-curdling ferment* which is abundantly found in some, especially sucking, animals, converts the soluble caseinogen of milk into the less soluble casein, which is precipitated in more or less fine particles. This change is analogous to the clotting of blood, and, like this process, requires the presence of calcium salts. The action of rennet thus differs from that of acids, which simply precipitate the casein, or, more properly speaking, the caseinogen or precursor of casein. As this occurs in all cases before milk is digested by ferments (either by pepsin, by pancreatin, or by papain) it is probable that the physical condition of the casein in finely divided particles, and perhaps the molecular change it undergoes, is that which is necessary for the rapid and efficient digestion of the milk; and a little consideration shows that this is so, since the precipitation of casein, and with it the fat, separates the milk into a liquid part containing the salts and lactose, more readily manipulated than the heavy milk itself; and a solid part (casein and fat), the casein particles of which are readily digested by the pepsin-hydrochloric acid, thus setting free the fat.

Although the hydrochloric acid of the gastric juice is in sufficient quantity during full digestion to precipitate the caseinogen of milk, the curdling ferment probably does this more effectually and earlier in the process of digestion, since a few seconds after milk is swallowed it becomes coagulated in the stomach, at a period before sufficient acid has been secreted for precipitation of the caseinogen.

A lactic acid ferment has been described by Hammarsten which changes lactose into lactic acid. It is probable, however, that this ferment comes from the bacteria present in the food, and is not a secretion of the gastric glands.

Cane sugar in the stomach is slowly transformed into grape sugar, an action ascribed to the agency of the mucus of the stomach. But such a transformation, although of great importance in digestion and nutrition, occurs only to an insignificant extent in the stomach, and is one of the chief functions of the succus entericus in the small intestine.

Action of the Pepsin-Hydrochloric Acid.—The action of the two principal agents of the gastric juice is solely on the albuminous or proteid constituents of the food and on gelatin and other albuminoids. At a temperature of between 35° and 40° C. pepsin in the presence of a 0·2 per cent solution of hydrochloric acid digests all proteids, transforming them into more soluble products (albumoses and peptones) in order to facilitate their absorption. An early stage of the transformation is into syntonin or parapeptone, a body closely allied to acid albumins, since it is soluble only in acid solutions, from which it is precipitated by neutralisation; and once in the precipitated form it is coagulated or rendered insoluble by heat. One of the next class of products, viz. hetero-albumose, is somewhat allied to syntonin since it is not soluble in water, but only in solutions of neutral salts (such as sodium chloride or magnesium sulphate) and in dilute acids or alkalies. It is in part precipitated from these solutions by removal of the neutral salts or by neutralising the solution, or by boiling the solution in neutral salts. The other albumoses, viz. proto- and deutero-albumose, are much more soluble products, since no salts, acids, or alkalies are requisite to hold them in solution, and they are not precipitated by boiling the solution: peptone has the same properties. From a practical point of view, as when the stomach contents require to be examined chemically, the chief digestive products to be sought for are proto- and deutero-albumose and peptone. In normal digestion in the stomach peptone does not appear to be found in great quantity; or it would be more accurate to say that an examination of the stomach contents during full digestion does not show the presence of a large quantity of peptone. This may be, as will be seen, because as soon as it is formed it is absorbed by the mucous membrane.

The chemical reactions of the bodies under consideration show that deutero-albumose is a product intermediate between proto-albumose and peptone, on account of its great solubility. Thus all these three bodies are soluble in distilled water, cold or boiling. Proto-albumose is precipitated by nitric acid in the cold, the precipitate being soluble in excess of the reagent and also soluble on warming, coming down again in cooling. Deutero-albumose is not so precipitated unless a certain quantity of sodium chloride or other neutral salt is present. Peptone is not precipitated at all by either method. Proto-albumose and deutero-albumose are both precipitated by saturating the solution with neutral ammonium sulphate; peptone is not so precipitated. All three bodies give a rose-pink colour when a trace of solution of sulphate of copper (1 per cent) and an excess of potash solution are added; this is the biuret reaction. (See also Examination of the Stomach Contents, Chapter V.)

The products of digestion just considered are formed from so-called "native" proteids, those, namely, which exist normally in the tissues of animals or plants, such as the myosin of flesh, the albumin of egg, the casein of milk, the gluten of bread, the globulins of plants. This class constitutes the most nutritious group of proteids. But there are other allied bodies which are not so useful as food, and which are best called "albuminoids." These are gelatin, chondrin, and elastin, which undergo digestion in the stomach; and keratin, which undergoes no change.

The kind of transformation of these bodies is the same as that previously described. The final digestive product from gelatin, the most important member of the group, is called gelatin peptone, the precursors of which during digestion are allied to the albumoses and are called "gelatoses." Gelatin peptone differs in reactions from proteid peptone, giving no reddish precipitate on boiling with Millon's reagent, and a slight yellow colour (not a deep yellow) on boiling with nitric acid. Chondrin, which is obtained from cartilage by boiling, also yields digestive products similar to those yielded by gelatin; and during digestion elastoses are formed from elastin. Gelatin is, however, the most important member of this group, since it is the body present in the boiled connective tissue of animal foods.

The Condition of Proteid Food as it leaves the Stomach.—All the proteid matter taken into the stomach as food is not digested there; the fluid which is passed through the pylorus into the duodenum contains the various proteid products of digestion (syntonin, albumoses, and peptone); and the syntonin is immediately precipitated with the pepsin by the alkaline liquids (bile and pancreatic juice) in the small gut. Although in human digestion a large part of the proteid food is left for the pancreatic juice to digest in the small gut, yet the action of the gastric juice is a good preparation for pancreatic digestion—just as the action of the saliva on the starch in the mouth, although inhibited in the stomach, is a good preparation for the completion of the digestion by the amylopsin of the pancreatic juice. A little consideration will make clear this useful action of the gastric juice. Most of the proteids taken as food are in the cooked form, that is more or less coagulated and insoluble. The gastric juice acts on this insoluble proteid, causing it to swell up and eventually to enter into solution. In this soluble form it is more easily digested by the pancreatic juice than in the insoluble. This is one action, but digestion by the gastric juice is certainly useful in another way. The proteids taken as food are of various kinds: animal myosin in flesh, albumin of eggs, casein of milk, gluten of flour, globulin and albumoses of plants and seeds—all with certain definite characteristics, and all possessing, presumably, a slightly varying molecular constitution. They have all to be transformed into the proteids of the human organism, and primarily it is supposed into the proteids of the blood—albumin and globulin. That they must undergo this transformation is shown by the fact that they are capable of supporting the nitrogenous metabolism of the body; that is, that the nitrogen they contain is used to nourish the tissues. This being so, it must be allowed that one action of the pepsin-hydrochloric acid of the gastric juice is to prepare these various proteids for entering the blood, to so alter their constitution that they can readily be transformed into the proteids of the blood; and the action of the pancreatic juice may be essential to complete this process.

Absorption in the Stomach.—Most of the digested food is absorbed in the small intestine; active absorption, however, takes place in the stomach—of sugar, salts, and proteids.

The *sugar* absorbed is the maltose formed by the saliva from starch and the small amount of glucose formed in the stomach by the inversion of cane sugar. During the absorption of maltose it is transformed into glucose, since the former is not found in the blood or the liver.

Some portion of the soluble *salts* is absorbed in the stomach, but there is probably an active interchange between the salts of the blood and those in the gastric contents during digestion.

The *proteids* are absorbed chiefly as albumoses and peptone.

Some proteids may be absorbed unchanged, as in the case of a large quantity of eggs being taken as food, egg-albumin appearing in the urine. Proteids in solution which are injected into the rectum, where there is no digestion, have also been proved to be absorbed unchanged. The absorption of proteids by the stomach in the form of albumoses is the ordinary mode, and one of the advantages of the action of the gastric juice is that it transforms the insoluble or colloid proteids into soluble and diffusible proteids—albumoses and peptone. The process of absorption of these digestive products is not purely mechanical, not simply a question of the diffusion of liquids, for in passing through the mucous membrane, whether of the stomach or the intestine, albumoses and peptone disappear as such. This was shown by Hofmeister, who also demonstrated that the stomach of an animal, removed from the body and kept in an appropriate warm chamber, could also cause this transformation of albumoses and peptone. Into what body they are changed has not yet been demonstrated; and although in several series of experiments performed by various observers, it has been assumed that "serum albumin" is the body formed, there is as yet no chemical proof that this is the case, and these observers use the term "serum albumin" loosely for the proteids of the blood-plasma, which contains serum globulin and fibrinogen as well as serum albumin. Most physiologists consider that the regeneration of albumoses and peptone is due to the lining epithelium of the alimentary canal. It is, however, certain that during absorption the albumoses and peptone disappear in their passage through the mucous membrane, and that they are not found in the blood stream or in the tissues of the healthy animal. Hofmeister some years ago stated that he had found these bodies in the blood during digestion, but the application of more recent methods by Neumeister has shown that this is not the case. If absorbed as such the albumoses would be inimical to the human body,

The question of the absorption of *water* in the stomach is one which has been the subject of much discussion and frequent investigation. It was until quite recently considered that water was absorbed in large quantities, although this conclusion would be a somewhat anomalous one, since very little water is actually absorbed in the small intestine, and the rationale of its absorption in the stomach could not be very evident. Tappeiner[1] came to the conclusion from his experiments that very little water was absorbed by the stomach, but that alcohol was readily taken up, and all substances soluble in it. His conclusion that peptone and glucose were not absorbed in any quantity by the stomach was shown to be erroneous by the experiments of von Anrep. Von Mering[2] showed conclusively in dogs that water given to the animal to drink flowed through the pylorus in more or less rhythmic gushes; and that the *whole of the water* drunk passed through the pylorus in this manner. Very little water is retained in the stomach when it is taken by itself. Such an experiment as this does not quite imitate the conditions in the stomach when a mixed meal with water is taken. In this case the bulk of the food and water is retained in the stomach for a considerable time, and absorption of water may take place, but probably not to any great extent. Whatever amount of water is absorbed with the soluble salts, sugar, and peptones is in all probability returned by the blood to the stomach contents. Von Mering also showed that carbonic acid in solution is readily absorbed by the stomach, as well as alcohol, glucose, cane sugar, lactose, and maltose. Dextrin and peptone are not really absorbed. He confirms Tappeiner's observation already referred to, viz. that alcoholic solutions of the substances mentioned are more readily absorbed than aqueous, a fact of some dietetic importance.

The non-absorption of liquid by the stomach is, as will be seen, an important factor in certain pathological conditions. It is also of physiological importance since the abstraction of water leads to a delay of digestion in the stomach and in the small intestine.

Process of Digestion of the Mixed Food in the Stomach.—

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[1] "Ueber Resorption im Magen," *Ztschr. f. Biol.*, München, Bd. xvi. p. 497, 1881.

[2] "Ueber die Functionen des Magens," *Verhandl. des XII. Congress f. innere Medicin zu Wiesbaden*, 1893. See also Edkins, *Journ. Physiol.*, Cambridge, vol. xiii. p. 445.

The chemical processes of digestion in the stomach have been discussed; but of greater importance in its relation to disease is the change undergone by the mixed food. The stomach is the receptacle of all the food taken, of the carbohydrates partially digested by the saliva, of the fats surrounded by tissue, of the proteids and of the salts. Although only the proteids are digested in the stomach, yet other changes occur which must of necessity be discussed.

To make the subject clear it will be considered under the following headings:—

1. The naked eye changes in the stomach during the digestion of a meal.
2. The effect on the mixed food of the digestion of proteids by the gastric juice and of the movements of the stomach.
3. The presence of organic acids in normal digestion.

1. In the intervals of digestion the mucous membrane of the stomach is a light pink colour, and the organ is practically empty, containing a small amount of liquid, slightly alkaline and containing salts and a little mucus. As soon as food enters the organ, the mucous membrane becomes more vascular and the "glands begin to discharge a clear, transparent fluid, which continues rapidly to accumulate as aliment is received for digestion. This fluid is invariably distinctly acid" (Beaumont).

2. The food which enters the stomach to be acted upon by the gastric juice is the mixed and masticated food; it consists of proteids, carbohydrates, fats, and organic and inorganic salts. The proteids are in a more or less insoluble form, being partly coagulated during the process of cooking. The fibres of the meat, consisting of myosin, are not completely disintegrated by mastication or cooking, although their connection with each other is loosened by the effect of cooking on the interstitial connective tissue, which has the result of transforming collagen into the readily soluble and digestible body gelatin. Of the other chief proteid foods, eggs as eaten are composed of coagulated albumin, globulin, and fat; milk, of caseinogen in solution mixed with fat and lactose; bread, of

gluten and albumoses mixed with carbohydrates—starch and dextrin. The starch and dextrin have been more or less dissociated from the other constituents of the mixed food by the action of the saliva in the mouth, so that when the carbohydrates enter the stomach they are in part already transformed into dextrin and maltose. The action of the swallowed saliva can only continue for a short time in the stomach, viz. from 15 to 45 minutes, as the increasing acidity and the action of the pepsin destroy the activity of the ptyalin.[1]

The process of digestion of this mixed food in the stomach is twofold. As soon as the food enters the organ, the movement of the walls begins, resulting in a very intimate and continuous mixing of the food, since this not only traverses the organ from the cardia to the pylorus along both curvatures and back again down the middle (Brinton), but is also worked towards the centre of the organ by the churning movement. Brinton's hypothesis regarding the course of the food does not accord with the actual observations of Beaumont (*op. cit.*), or with the experimental results of Hofmeister and Schütz[2] in their research on the automatic movements of the dog's stomach. Both Beaumont and Hofmeister and Schütz concluded that the food travelled along the greater curve and back again along the lesser. From the position and shape of the stomach this conclusion would appear probable. The movements of the stomach not only intimately mix the food, but reduce the size of the masticated particles and cause the gastric juice secreted by the mucous membrane to act on all the particles swallowed. The chemical changes which begin as soon as the secretion of gastric juice are at first slight, owing to the slight acidity of the stomach contents; the first portion of acid secreted being utilised in neutralising the swallowed saliva as well as the food if it is alkaline. But in about an hour or an hour and a half from the ingestion of a large meal, the acidity of the stomach rises to 0·2 per cent or even higher. The hydrochloric acid forms a loose compound with the proteids present. This loosely combined acid does not give all the reactions of the free acid. Proteids can only take up a certain proportion of acid, and therefore as digestion proceeds the acidity due to hydrochloric acid is produced both by the loosely combined acid and by the free acid. The effect on the mixed food of the digestion of the proteids by pepsin-hydrochloric acid leads to a great disintegration of the food, although it is not a complete digestion. Thus with meat the gelatin of the cooked connective tissue is readily digested, loosening the fibres of the meat, which also swell up and are partially digested. Any fat present in the meat is also separated by this digestion, and is melted by the temperature of the body. With eggs, the proteids are caused to swell up and are partially digested, while the fat is freed and melted. With milk, the casein is first precipitated with most of the fat, which is, however, set free as the digestion of the casein-precipitate proceeds. Thus, although one of the chief effects of the pepsin-hydrochloric acid is the digestion of proteid substances, yet another and not less important result is the disintegration of the food whereby the fat is separated, and, being melted by the body temperature, floats on the surface of the gastric contents, and the proteids and carbohydrates which are partly in solution are by their fine subdivision in a very suitable condition for rapid digestion by the pancreatic juice and bile in the small intestine. To the mixed food as it is digested by the stomach—consisting as it does of particles of undigested proteid and carbohydrate and melted fat floating on an acid liquid, containing in solution, besides salts and organic acids, the products of digested proteids (syntonin, albumoses, and peptone), and some maltose and unaltered cane sugar and lactose—the term “ chyme ” is applied.

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[1] See Langley, *Journ. Physiol.*, Cambridge, vol. iii. p. 246 ; Langley and Eves, *ibid.* vol. iv. p. 18 ; Chittenden, etc. quoted by Halliburton, *op. cit.* p. 628.

[2] F. Hofmeister and E. Schütz, "Ueber die automatischen Bewegungen des Magens" ; *Arch. f. exper. Path. u. Pharmakol.*, Leipzig, 1885, Bd. xx. p. 1.

Although during digestion the stomach is a closed bag, yet the pylorus dilates from time to time as the contents become more liquid, and allows the extrusion into the duodenum of small quantities of the acid liquid contents. This action, which is not a regularly rhythmic one, but occurs at irregular intervals, is of great service, as, instead of a large quantity of acid liquid being propelled into the duodenum for it to cope with, small quantities entering it are readily neutralised by the bile and pancreatic juice, the pepsin and syntonin being precipitated. Thus, while the stomach is preparing the food by partially digesting the proteids and by trituration and mixing of the food, the pancreatic juice and bile are already finishing the digestion of the carbohydrates and proteids. This process of preparation of food in the stomach and its gradual passing on to the duodenum for completion of digestion is a very important one, and irregularity in its performance probably forms a fruitful source of disease. If a small quantity of liquid be swallowed with the food, very little leaves the stomach until digestion is completed. With a large quantity of liquid, some soon passes through the pylorus. The fat separated from the mixed food in the stomach is the last portion of food to leave the organ, probably solely because it floats on the stomach contents.

3. The consideration of the changes in the food during digestion would be incomplete without a discussion of the effect of the organic acids and salts which are taken with the food. Thus meat as eaten contains sarcolactic acid; fruits and vegetables contain, either free or combined, citric, acetic, and tartaric acids, and vinegar (acetic acid) is often mixed with food. The salts of these acids are decomposed by the hydrochloric acid of the gastric juice, so that the acid is set free and the acidity of the stomach contents increased. Moreover, after the entrance of carbohydrate food into the stomach, it is stated that lactic acid is formed by bacterial decomposition of the carbohydrates, and according to Ewald, it is the chief acid in the stomach for the first three-quarters of an hour of digestion under the circumstances mentioned.

Ewald and Boas[1] investigated the process of digestion in three young individuals, two with normal digestion and one with vomiting neurosis. They found that ten minutes after giving 60 grammes (about 2 ounces) of white bread, lactic acid was contained in the stomach contents, and that the quantity increased for 30 or 40 minutes. Hydrochloric acid was then found, and this increased while the lactic diminished, until it disappeared. After meat a similar condition was noticed, viz., first, the appearance of sarcolactic acid, then in 60 to 90 minutes both this acid and hydrochloric acid, and in 100 to 120 minutes hydrochloric acid alone. Fish gave the same results as meat, and with potatoes an appreciable quantity of lactic acid was found. None was found when egg-albumin was given as food.

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[1] *Virchow's Archiv*, Bd. ci. p. 325.

Cahn and von Mering[1] obtained the following results in three healthy individuals, two of whom were on milk diet, and one on a pure meat diet:—

	Period, after eating, of removal of stomach contents.	Percentage of HCl.	Percentage of lactic acid.	Percentage of volatile acids.
Case 1. Healthy male, aged 20, on pure meat diet	3½ hours	0·204	0	0
Case 2. Healthy male, aged 20, on milk diet	½ hour	0·058	0·101	0·003
Case 3. Healthy male, aged 36, on milk diet	1 hour	0·034	0·02	0·009
	2 hours	0·164	0·281	0·075

It is evident, therefore, that even in healthy individuals on a milk diet, that a certain amount and in some cases a large amount of lactic acid may be present in the stomach contents during digestion. With a meat diet, the acidity of the stomach contents was found to be due almost solely to hydrochloric acid, a result apparently antagonistic to that of Ewald and Boas. Meat does contain sarcolactic acid, and it is apparent that this will be set free in the stomach contents during the process of digestion; it will therefore be found in the early stages of gastric digestion, and would then either be absorbed, or would combine with the chlorides as suggested by Maly. At any rate, it disappears from the stomach as digestion proceeds, and it is therefore not surprising that Cahn and von Mering did not find any lactic acid present in the stomach three and a half hours after a meal of meat alone. The presence of lactic acid after a meal of bread was explained by a decomposition of the carbohydrates of the bread occurring in the stomach by means of bacteria. There is no necessity to suppose that this occurs, since during the preparation of bread, not only acetic but lactic acid is formed. Acetic acid is a further product of the action of yeast (saccharomyces cerevisiæ) on carbohydrates and the alcohol formed from them. The acetic acid fermentation may occur, and lactic acid and butyric acid are also formed by the action of the lactic acid and butyric acid bacilli. The acidity of bread and of biscuits increases with keeping, even for one or two days.[1] It is not therefore at all surprising that after a diet of bread alone lactic acid should be found. The presence of lactic acid in this case has nothing to do with the functions of the stomach; it is simply an acid taken in with the food. And this is shown to be the case by the experiments of Martius and Lüttke[2] and of Boas,[3] for when food which does not contain lactic acid is given, this acid is found only in traces in the stomach contents. With a milk diet in healthy adults, this question is somewhat different, as shown by Cahn and von Mering's results. In this case the percentage of lactic acid in one individual half an hour after food was 0·101, and in another individual it was in one hour after food 0·2, and in two hours 0·281 per cent. Here there is evidence of distinct formation of lactic acid in the stomach in normal digestion, and the question is how is it formed. There is no evidence to show that in the digestion of milk by pepsin-hydrochloric acid, lactic acid is formed; therefore, it must be concluded that it is formed by fermentation of the lactose of the milk, a fermentation by means of the lactic acid-producing micro-organisms swallowed with the food. It is perhaps not wise to assume from these two experiments of Cahn and von Mering's that lactic acid is always found in quantity when milk is taken as a food, since there is no account given of the milk taken (whether boiled or not) and no account of the individuals except that they were healthy. It will be seen that many bacteria have been found in the stomach capable of forming lactic acid from carbohydrates (p. 73).

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[1] *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxxix. p. 233.

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[1] See König, "Die menschlichen Nahrungs- und Genüss-Mittel," Bd. ii. p. 615, 3rd ed., 1893.

[2] "Die Magensäure des Menschen," 1892, p. 55.

[3] "Diagnostik u. Therapie der Magenkrankheiten," 1894, 3rd ed., part I. p. 32.

Lactic acid does not, however, play any great part in normal digestion: it was at one time considered the chief acid in the stomach. And although peptic digestion will take place in the presence of lactic acid and even of nitric acid, it was conclusively proved by Bidder and Schmidt, and confirmed by subsequent observers, that the acid secreted by the gastric glands and acting with pepsin as the agent in digestion is hydrochloric acid.

In artificial digestion experiments, it is found that gastric digestion proceeds most rapidly in the presence of hydrochloric acid, less rapidly in the presence of acetic or lactic acid, and scarcely at all when butyric acid is the only acid present. These acids all form acid albumin when they are allowed to act on uncoagulated egg-albumin, but with varying degrees of facility. Hydrochloric acid is the most active in this respect, butyric acid the least active, while acetic and lactic acids occupy an intermediate place. The effect of these acids on peptic digestion is shown by the following experiment:—

Into each of eight tubes, 50 cc. of a solution of egg-albumin were placed and coagulated by heat; 50 cc. of water were added to each. The tubes were divided into sets of two, and 0·2 per cent HCl added to one set, 0·2 per cent acetic acid to another, 0·2 per cent lactic acid to a third, and 0·2 per cent butyric acid to the fourth. Four tubes were set aside as controls, and to each of the remaining four, 4 cc. of an active solution of pig's pepsin were added. All the tubes were then placed in the incubator at 95° F. for 1 hour 40 minutes; and the amount of digestion taking place was estimated by comparing each tube with the control, by estimating the amount of coagulated albumin undissolved, by the amount of precipitate on neutralising and by the intensity of the biuret reaction for peptones. The result is shown in the table on the next page.

From this experiment it is quite clear that in the presence of acetic and lactic acids, pepsin is much less active than when hydrochloric acid is alone present; and that lactic is, if anything, less active in hindering peptic digestion than acetic acid. With the amount of butyric acid used, no digestion whatever occurred, and there was no formation of acid albumin in the absence of pepsin.

TABLE SHOWING THE ACTIVITY OF PEPSIN IN THE PRESENCE OF HYDROCHLORIC, ACETIC, LACTIC, AND BUTYRIC ACIDS. *Digestion at 95° Fahr. for 1 Hour 40 Minutes.*

Composition of mixture.			Result.			
			Amount of albumin undissolved.	Biuret reaction =peptone.	Precipitate on neutralisation.	General result.
Tube 1.	Coagulated albumin Water . . . **HCl** . . . Pepsin solution .	. 50 cc. . . . 50 cc. . . . 0·2 per cent . 4 cc. . .	Nil.	Brilliant.	Opalescence.	**Active digestion.**
Tube 2.	Control of (1) .	. Same but no pepsin	Nearly all.	Nil.	Slight precipitate.	Small amount of acid albumin formed.
Tube 3.	Coagulated albumin Water . . . **Acetic acid** . . Pepsin solution .	. 50 cc. . . . 50 cc. . . . 0·2 per cent . 4 cc. . .	0·130 gramme, dried at 100° C.	Faint.	Slight precipitate.	**Slight digestion.**
Tube 4.	Control of (3) .	. Same but no pepsin	Nearly all.	Nil.	Slight precipitate.	Small amount of acid albumin formed.
Tube 5.	Coagulated albumin Water . . . **Lactic acid** . . Pepsin solution .	. 50 cc. . . . 50 cc. . . . 0·2 per cent . 4 cc. . .	0·079 gramme.	Very faint.	Moderate amount of precipitate.	**Slight digestion.**
Tube 6.	Control of (5) .	. Same but no pepsin	Nearly all.	Nil.	Slight precipitate.	Small amount of acid albumin formed.
Tube 7.	Coagulated albumin Water . . . **Butyric acid** . Pepsin solution .	. 50 cc. . . . 50 cc. . . . 0·2 per cent . 4 cc. . .	0·158 gramme.	Nil.	Very slight opalescence.	**No digestion.**
Tube 8.	Control of (7) .	. Same but no pepsin	Nearly all.	Nil.	No precipitate.	No acid albumin formed.

In other experiments, it was seen that in the presence of hydrochloric acid and of one or other of the acids mentioned, the activity of pepsin was diminished by butyric acid, but not to a great extent by lactic acid, as compared with a digestive mixture in which hydrochloric acid alone was present.

It may be safely concluded, therefore, that when the stomach contents are hyperacid from the presence of organic acids, pepsin can have but little activity; but that the presence of a small percentage of lactic or acetic acid in association with hydrochloric acid (such as commonly occurs in the normal digestion of mixed food) is no great hindrance to the chemical processes (see Chapter IV.).

The organic acids, whether taken in with the food or formed from carbohydrates in the stomach, play an important *rôle* in the dietary, since they are a source of carbonates, thus preserving the alkalinity of the blood and tissues; they are also antiscorbutic agents. Their excessive ingestion or formation in the stomach leads, as will be discussed later, to great functional disturbances of digestion.

Relation of Digestive Processes in the Stomach to Salivary and Intestinal Digestion.—Although the process of digestion in the stomach is in itself an entity, yet the efficient action of the saliva is a very necessary precursor to normal digestion; for not only does the saliva aid mastication by moistening the food, but it rapidly digests the cooked starch in the mouth, and acts for a short time in the stomach itself until the acidity rises to a degree sufficient to stop the action. The alkalinity of saliva is equal to 0·097 per cent of sodium carbonate (Na_2CO_3), and its action is stopped by 0·003 per cent of hydrochloric acid (Chittenden and Smith).[1] According to these observers, therefore, the salivary diastase could act in the stomach for only a very short time. Of more importance than the action of the saliva is the change which the digested food undergoes when it leaves the stomach. It then consists of a pea-soupy mass, with an acidity due mostly to hydrochloric acid, but partly also in some instances to organic acids, such as lactic and acetic acids, and containing in solution pepsin, some peptones, maltose and salts, and containing also shreds of undigested food, such as muscle fibre and particles of albumin, starch grains, and globules of fat. When this acid mass meets the alkaline bile and pancreatic juice, the acidity is more or less neutralised, so that the liquid becomes alkaline, or neutral, or remains for a time slightly acid. The pepsin is precipitated and rendered inactive, while the hydrochloric acid of the gastric contents has, during the process of neutralisation, combined with the sodium of the bile salts and of the carbonate of sodium, and set free the bile acids. During this change the contents of the small intestine become alkaline, and remain so during the whole period of their stay in the small gut; but this does not appear to be so in all cases, and the contents may remain acid, although only slightly so.[1] The acidity of the contents in the lower part of the small intestine is probably due to lactic acid—possibly, in some cases, to butyric acid, both of which are the products of bacterial fermentation of the carbohydrates. A large amount of lactic acid is, however, not formed. The growth of bacteria in the small intestine is hindered to some extent by the setting free, in the first instance, of the bile acids, which are antiseptic in action; and although there is some lactic acid fermentation of the contents, yet there is normally no putrefactive decomposition of the proteids. The contents of the small intestine have, during normal digestion, no putrefactive odour. There can be little doubt that the digestion in the intestine is affected, at least in its early stages, according to the degree of acidity of the chyme as it enters the duodenum. The chief condition appears to be that if the chyme is very acid from hydrochloric acid, the digestion of starch in the intestine is greatly delayed (Boas); [2] but this delay cannot last any length of time, since the acidity of the contents is being neutralised by the continued secretion of the bile and pancreatic juice.

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[1] *Trans. Connecticut Association*, 1885, vol. vi. p. 143. Abstract in Maly's *Jahresbericht*, 1886. The figure, 0·097, was obtained as the average estimate from fifteen specimens.

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[1] See Macfadyen, Nencki, und Sieber, "Ueber die chemischen Vorgänge im menschlichen Dünndarm," *Arch. f. exper. Path. u. Pharmakol.*, Leipzig, Bd. xxviii. p. 311, 1891.

[2] Boas, *Ztschr. f. klin. Med.*, Berlin, Bd. xvii. p. 155. Abstract in Maly's *Jahresbericht*, Bd. xx. p. 261.

CHAPTER II

DIGESTIBILITY OF VARIOUS ARTICLES OF DIET AND THE EFFECT ON DIGESTION OF FOOD ACCESSORIES (CONDIMENTS, ALCOHOLIC DRINKS, TEA, ETC.).

A KNOWLEDGE of the digestibility of ordinary articles of diet and of the effect on digestion of condiments and alcoholic and other drinks is essential for the proper treatment of disorders of the stomach, and as essential as a knowledge of the pathological changes in the movements and secretion of the organ. Individuals differ in their digestions as they do in their intellectual or emotional characteristics; and as regards the eating of food, each healthy man must to a great extent be a law unto himself. Observations have been made, however, which are of great importance in the dietary of the healthy as well as the sick. Speaking generally, the proper digestion of food, *i.e.* the digestion which after a meal goes on without any feeling of digestion, and with only a sense of general repose, depends on the following conditions, the movements of the stomach, the secretion of the gastric juice, and the absorptive processes being unimpaired :—

1. The food must be properly cooked or prepared.
2. The food itself must be a mixed food, consisting of proteids, carbohydrates, fats, and salts in certain proportions, too much of one constituent not being present.

3. The food must be digestible and must not contain a large quantity of indigestible matter; *e.g.* cellulose.
4. The food must not be too acid, and must not be mixed with too much or too little liquid.
5. It must be flavoured, but not mixed with too large a quantity of condiments or alcoholic drinks.

The Food must be properly Cooked or Prepared.—By cooking the food is rendered more digestible. This is especially the case with vegetable foods, as they are taken chiefly for the starch they contain: the cellulose coats of the starch grain during boiling are ruptured, causing the enclosed starch (granulose) to swell and to be set free. With baked vegetable foods some of the starch in the outer part is transformed into dextrin, a change which must be considered a great advantage to digestion. This is the case with bread during the baking; but in addition the flour is fermented, some of the starch being thus transformed into dextrin and sugar. Bread is really therefore a partially digested vegetable food.

With animal food, such as meat, the fibres are loosened by the transformation of the collagen of the connective tissue into gelatin, an easily digestible substance; but the cooked meat-fibre itself is not more digestible than the raw. There is no doubt another advantage in cooking animal foods, viz. that the coagulation and partial hardening of the proteid constituents renders them more easy of mastication, an essential process in good digestion.

Cooking, especially in meat, develops flavours, which are beneficial and indeed necessary, since unsavoury food leads eventually to loss of appetite. By making the food hot, animal heat is economised, since the food has not to be raised to the temperature of the body. As a rule there is a fall of temperature in the stomach during the early period of digestion, a fall ascribed to the secretion of gastric juice. By cooking, meat loses about 20 to 30 per cent in weight, and vegetables gain from about 3 to over 50 per cent, the starchy vegetable foods showing the greatest gain. The loss or gain in weight is due to the amount of water lost or taken up.

Cooking destroys to a great extent parasites in the food:

bacteria and worms. Bacteria are, however, by no means absent from the eaten food, as is shown by their abundance in the intestines.

2. The successful treatment of functional disturbances of digestion is so largely a question of diet that it is not out of place to consider here certain facts of first importance in the dietary of healthy individuals. The dietary of healthy individuals has not been determined by scientific investigation; it is the outcome of experience and custom; yet the scientific investigation of such empirical diets has led to the establishment of data which are true within certain limits. The diet of a healthy individual must consist of the following constituents: proteids, fats, carbohydrates, mineral salts, vegetable acids, and water. Proteids are essential to the organism, since they contain nitrogen in a form which after digestion is capable of being assimilated by the tissues. They are sources of muscular energy, as Liebig originally showed, and Pflüger and others have confirmed.[1] Fats and carbohydrates are of service, not only by their combustion maintaining the heat of the body and in maintaining muscular activity, but also from the fact that their presence in a diet up to a certain amount enables less proteid food to be taken, thus obviating the dangers of an excess of proteid in the diet. They are therefore termed proteid-sparing foods. Mineral salts and water are necessary for the solution of proteid bodies in the liquids of the body and for the formation of tissues (bone, etc.), sodium chloride and calcium phosphate being essential to every developing tissue. Vegetable acids, free or in combination, are of use as anti-scorbutics, and for keeping the tissues alkaline, since they form carbonates in the system. The correct way of regarding these food-stuffs is that they are essential for the maintenance of the activity and metabolism of the protoplasmic tissues; that as regards fat and carbohydrates, although they may be said to maintain heat and muscular activity, yet they do so by being metabolised by the tissues and not by any chemical combustion independent of the tissues.

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[1] For a summary of the recent work on this subject, see a paper by E. Starling, M.D., "Researches on Proteid Metabolism," *Science Progress*, April 1894, p. 141.

The amount of the food-stuffs which is requisite to maintain health has been variously estimated; but the diet calculated by Moleschott as answering for a man weighing 150 lbs. and doing a medium amount of work, may be taken as about the average. Thus, according to this estimate, a man would require 130 grammes (4·59 oz.) of proteid, 84 grammes (2·96 oz.) of fat, 404 grammes (14·26 oz.) of carbohydrates, 30 grammes (1·06 oz.) of salts, and about 2800 grammes (70 to 80 oz.) of water in each day to maintain health. This proportion of food-stuffs may be conveniently expressed in such a simple diet as the following:—

Article of food.	Weight in grammes.	Weight in oz.	Proteids. Weight in grms.	Fat. Weight in grms.	Carbo-hydrates. Weight in grms.	Salts. Weight in grms.	Water. Weight in grms.
Meat	385	13·5	82·8	21·0	—	3·85	279·0
Butter	50	1·7	0·5	42·2	—	0·60	6·6
Milk	500 cc.	17·7	17·7	18·4	24·4	3·50	435·8
Bread	280	9·8	22·0	4·2	137·7	3·64	112·0
Flour	200	7·0	24·0	3·7	137·3	3·56	26·7
Vegetables	225	7·9	4·5	—	11·2	2·25	202·2
Potatoes	130	4·6	2·7	0·2	27·30	1·43	97·5
Sugar	70	2·4	—	—	67·4	3·50	2·0
	1840	64·6	154·2	89·7	405·3	22·33	1001·8

To this diet common salt is added to make up the deficiency in sodium chloride and water drunk to make up the deficiency in water.

For a child from six to seventeen years of age the dietary is not so large as the foregoing. Such a child requires on the average 6 oz. of meat, 10·5 oz. of bread, 6 oz. of potatoes, 0·5 oz. of butter, 9 oz. of milk, 3·5 oz. of flour (for soup), and 6 oz. of various kinds of vegetables; but the amount of milk may be greatly increased, reducing the amount of meat.

In constructing diets to maintain health, it is important to remember that the end cannot be obtained unless the proteid constituents of the food bear the proportion to the non-proteid (*i.e.* fats and carbohydrates) of 1 : 3·5 to 4·5; and that the amount of fat must not be in greater proportion to the carbohydrates than 1 to 9. In the infant, the proportion of fat to the carbohydrates is as 1 : 1·4; in the adult in easy circumstances it is as 1 : 3·4; and in the workman as 1 : 5 or even less. The facts just mentioned are essential for the nutrition of the healthy organism, and although diet in disease is very different, yet as far as possible dieting must be on the lines mentioned (see Chapter XI.).

3. Of as great importance as the amount of food and food-stuffs necessary to maintain health is the digestibility of the food taken, and in disease this question of digestibility becomes of prime importance. Individuals may show an idiosyncrasy with regard to the digestibility of particular articles of diet, but of greater importance is the consideration of the digestibility of the various foods and food-stuffs as these differ among themselves, apart from any idiosyncrasy of the individual. The digestibility of the food-stuffs as investigated by Rubner may be stated in the following table, 100 being taken as a figure representing complete digestion :—

DIGESTIBILITY OF FOOD-STUFFS (PERCENTAGE DIGESTED).

	Meat.	Eggs.	Milk.	Cheese.	Rice.	Potatoes.	Peas.	White bread.	Black bread.	Carrots.
Proteid .	97·7	97	92	97	80	75·0	80	78	68	79·5
Fat .	80·0	95	95	95	—	—	—	—	—	—
Carbohydrates	—	—	—	—	99	92·5	95	99	88	82

It is thus seen that the proteids of animal foods (meat, eggs, milk, cheese) are far more digestible than those of vegetables (potatoes, peas, bread, etc.), and that the carbohydrates of rice and white bread are the more digestible.

The digestibility of various starches by the pancreatic diastase is shown in the following experiments. The starch was prepared in a pure state, and dissolved in boiling water. Each starch solution was of a known strength, and the diastase added was of the same quantity and strength in each experiment. The starches used were arrowroot (St. Vincent and Bermuda), the starch of fresh green peas and of pea flour, of wheaten flour, of rice, barley, and potatoes. The most digestible of these starches is in order, St. Vincent arrowroot, Bermuda arrowroot, and starch from fresh green peas. Then come in order, starch from wheaten flour, from potato, from rice and from barley, that from pea flour coming last. The experiments showed that a given amount of ferment could produce in a certain time a greater amount of sugar (maltose) from arrowroot (St. Vincent and Bermuda) than it could from the starch from green peas and the other starches: that from the starch of wheaten flour and of potatoes about the same amount of maltose was formed, but much less from the starch of rice or barley. These facts are of some importance in dietetics, and in great part explain the constipating action of arrowroot, since the more digestible a food is the more constipating it is.

As regards the digestibility of fats, but little need be said; all the important fats in a dietary are from the animal kingdom, the fat of meat and that of milk (butter), the latter being the more easily digested.

It must always be borne in mind, however, that the digestibility of the food-stuffs is only a small part of the question of the digestibility of food since the stomach is the receptacle of the mixed food, and the length of time the different articles of food remain in the organ is of great importance. This is especially so in disease, since so many of the disorders of digestion arise from delay of food in the stomach. The following table shows the results of the observations in two healthy individuals with gastric fistula:—

TABLE OF THE DIGESTIBILITY OF ARTICLES OF DIET IN THE STOMACH.[1]

Food.	Mode of preparation.	Length of time the food remained in the stomach till digested, absorbed, or discharged.	
		Beaumont.	Richet.
Schnaps	...	...	30–40 m.
Milk	...	...	30 m., 1 hr.
Rice	boiled	1 hr.	
Peas, with bacon fat	...	...	1–2 hr. 30 m.
Baked potatoes	...	...	1 hr., 2 h. 15 m., 2 hr. 30 m.–3 hr.
Eggs, whipped	raw	1 hr. 30 m.	
Barley soup	boiled	1 hr. 30 m.	
Salmon trout	boiled	1 hr. 30 m.	
Meat	...	...	1 hr. 30 m., 2 hr. 30 m., 4 hr., 5 hr. 30 m.
Sago	boiled	1 hr. 45 m.	
Spinach	...	...	1 hr. 45 m., 2 hr., 4 hr.
Tapioca	boiled	2 hr.	
Barley	boiled	2 hr.	
Milk	boiled	2 hr.	
Fresh eggs	raw	2 hr.	
Cabbage, with vinegar	raw	2 hr.	
Soup, with fat and bread	boiled	...	2 hr.
Rice, with fat	...	...	2 hr., 2 hr. 45 m., 3 hr., 3 hr. 15 m.
Milk	unboiled	2 hr. 15 m.	
Fresh eggs	roasted	2 hr. 15 m.	
Ox-liver	raw	2 hr. 15 m.	
Gelatin	boiled	2 hr. 30 m.	
Lamb	broiled		
Hash—meat and vegetables	warmed		
Beans	boiled		
Potatoes	boiled or roasted		
Cabbage	boiled	...	2 hr. 30 m., 3 hr. 45 m.
Macaroni and fat	boiled		
Eggs	soft-boiled		
Beef-steak	...	3 hr.	
White bread	baked		
Ham	boiled		
Lean beef	roasted		
Fish	boiled		
Mutton	broiled, boiled or roasted		
Pork	roasted	3 hr. 15 m.	
Poultry	roasted	4 hr.	
Veal	roasted		
Brown bread	baked		
Pork	salted	5 hr.	
Eggs	hard-boiled		

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[1] Beaumont, "Experiments and Observations on the Gastric Juice and the Physiology of Digestion," Edinburgh, 1838 (reprint). Richet, "Du Suc gastrique chez l'Homme et les Animaux," Paris, 1878.

It is clear from this table that from the experiments in these two individuals in whom digestion was normal, the following conclusions may be drawn :—

1. That the flesh of animals remains from two and a half to five hours in the stomach, the most digestible being lamb, and then beef-steak, mutton, veal, and pork, while fish is as digestible as mutton.

2. Starchy foods (rice, tapioca, barley) remain about two hours in the stomach; beans, peas and potatoes and bread remaining rather longer.

3. Even for the same food-article there are varying times during which it remains in the stomach, showing that the functional activity of the stomach varies at different times.

Effect of an Excess of Food.—The time during which food remains in the healthy stomach depends to a great extent on its bulk and its digestibility. The healthy stomach can cope with a large amount of food taken at one meal, and digest it with little or no disturbance. Disturbances of digestion arise, as will be seen, when very large meals are the rule and not the exception. Feeding a healthy man with a test meal, consisting of soup, a "large" beef-steak and a roll of bread, Leube found that the stomach was quite empty in seven hours, only a clear liquid with a few flocculi of mucus being removed by the sound. Such a meal as this is no doubt physiologically correct for the largest meal in the day ; smaller meals will remain a shorter time in the stomach. If such a meal or its equivalent be repeated during the day three times, at 9 A.M., at 1 P.M., and at 7 P.M., it is evident that the stomach will never be empty, will never be resting. Kretschky, for example, found in a patient with a gastric fistula that the time of digestion of the breakfast was four and a half hours; of dinner, seven hours; and of the evening meal seven to eight hours.[1] Although it is a matter of common observation that individuals vary greatly in the amount of food they can eat with impunity, yet few individuals can eat three large meals at intervals of four to six hours during the day without sooner or later suffering from the results of excess of food.

Physiological Results of an Excess of Food.—Food taken in excess of the actual requirements of the body leads to corpulence or to disorders of digestion, the gain in weight being chiefly in the form of fat. If the proteid constituents of the diet are in excess, the organs of digestion are overworked, and the nitrogenous tissues of the body, especially the liver and muscles, are also overworked. It must be remembered that the changes in the nitrogenous tissues result in one direction in the production of a certain amount of urea, and the amount of nitrogen in this substance has to be made good by the nitrogen taken in as food, in order that the nitrogenous equilibrium of the body may be maintained. An excess of proteid food in the dietary increases the amount of urea excreted, and thus the amount of work thrown on the nitrogenous tissues, especially the liver and muscles. In this way an excess of proteid food leads in all probability to gout, for uric acid, which is one of the features of that disease, must be derived from the tissues, and is formed by a physiological process similar to the formation of urea.

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[1] Kretschky, *Eulenberg's Real-Encyclopedia*, vol. iv. p. 241.

Proteid substances are to some extent a source of fat in the body when they are mixed with the other food-stuffs in the proper proportion of a diet (Voit); but if the proteid food-stuffs are greatly in excess of the carbohydrates and fat, then the body uses up its store of fat, since proteids cannot replace the fat in a dietary. In this way, *i.e.* by restricting the diet mainly to proteid substances (as meat) the body wastes, as in Banting's treatment of obesity. Pflüger, however, from his recent experiments has concluded that in no case can fat be produced in the body from the proteid of the food: and that if proteid is retained in the body, it is retained as such and not as fat.[1]

The physiological effect of an excess of fat in the diet is an increase of the deposit of fat in the body, and if the excess be great a diminution in the digestion of fat, which leads to a decomposition of the fat in the intestines. The amount of fat requisite in an average diet for the maintenance of health is a little under 3 ounces, which would be represented in a dietary by the fat in about $1\frac{3}{4}$ ounces of butter, in $17\frac{1}{2}$ ounces of milk (containing $\frac{3}{5}$ ounce of fat) and in $13\frac{1}{2}$ ounces of meat (containing $\frac{3}{4}$ ounce of fat). If more than this amount is taken the fat is passed in the motions either undigested, or in the form of fatty acids in combination, chiefly with calcium.

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[1] See Starling, *Science Progress*, April 1894, p. 145.

An excess of carbohydrates in the diet leads, like that of fat, to corpulency, but its chief physiological importance is associated with the fact that the amount of indigestible matter taken with the food is greatly increased. This indigestible matter is chiefly cellulose, which is present in great amount in ordinary vegetables, but is also present in bread, especially in the coarser forms of bread, such as black bread, brown bread, and whole-meal bread (see also p. 60). Cellulose is not digested in the human alimentary canal, although in that of the herbivora (cows, horses, etc.) a certain and probably considerable amount of it is digested by means of bacteria and utilised by the body. Its admixture with the food of man diminishes the amount of food digested and absorbed. Thus it was found by Schuster that in a prisoner on a vegetable diet with 104 grammes of proteid in the daily diet, an amount of nitrogen equal to 70 grammes of proteid was passed in the fæces, while in a prisoner on a mixed diet of bread, vegetables, milk, and meat, with 87 grammes of proteids in the diet, an amount of nitrogen equal to only 30 grammes of proteids was found in the fæces.

An excess of vegetable food in the diet produces copious, soft and watery fæces; an excess of animal food produces scanty and tenacious fæces.

The difference in the dietary of the workman and those more well to do is that the workman takes his nitrogenous food (proteid) chiefly in the form of vegetable products, there being an excess of carbohydrates in the diet, while the better classes take most of their proteid food in the form of animal products. Thus in a particular instance a workman took 27 per cent of his proteids in the form of meat, while a young professional man took 65 per cent (Förster).

4. The effect of a large amount of salts (organic and inorganic) and of free acids taken with the food is an important consideration in dietetics. Vegetable food is rich in

salts, chiefly phosphates of potassium, and an excess of potassium salts, besides their depressing effect generally, increases the excretion of sodium chloride, which is withdrawn from the tissues. The converse is also held to be true (Förster), viz. that an excess of sodium chloride in the diet increases the excretion of potassium salts. There is no doubt a very nice physiological balance between the sodium and potassium salts in the body, and it is thus possible that an excessive ingestion of either might lead to an increased excretion of both.

An excess of organic salts and acids (acetic, citric, malic, tartaric) leads to alkalinity of the urine, but as regards the stomach they lead to delay in the digestion of food: since whether taken as salts or as free acids (vinegar and lemon juice, and sour fruits) they tend to diminish the digestion of proteids in the stomach and to diminish the secretion of gastric juice (see also p. 31 *et seq.*).

An excess of liquid taken with the food also delays digestion. This is so not only in artificial digestion experiments, but also in the stomach. In artificial digestion experiments, pepsin acts more energetically if the liquid, although of the proper degree of acidity (0·2 per cent), is not in large amount. But in digestion in the stomach this dilution of the contents, by an excess of liquid, is of more importance; for although the healthy stomach can cope with a relatively large quantity of liquid taken with the food, which is more or less rapidly propelled into the duodenum, yet in certain cases of functional disorders of digestion, where there is not only defective secretion of gastric juice, but diminished movement, an excess of liquid taken with the food delays digestion, since it dilutes the pepsin-hydrochloric acid already present in the stomach. At a certain stage of digestion, dilution of the digesting mass aids the process. Thus in artificial digestion experiments, the action of the pepsin-hydrochloric acid on proteids stops after a time, but recommences if more liquid be added. This effect is expressed by saying that the action of the ferment becomes inhibited by an accumulation of its products. Such facts as these are important in connection with one dietetic rule for persons with functional disorder of digestion, viz. that liquids are best taken at the end of a meal or a short time after it.

The physiological purposes which liquid taken with meals serve may be considered as two in number: a certain amount of water (more than the food contains) is necessary for the efficient action of the gastric juice, and a certain amount of water is necessary for the absorption of the salts and digested products, and for facilitating the expulsion of the chyme through the pylorus into the duodenum.

5. *Effect of Food Accessories on Digestion.*—Civilised man, and for the matter of that savage man also, has his food flavoured. Many foods either possess a natural flavour or one becomes developed in them during the process of cooking; as in meat, for example. Flavouring agents are also added. Tasteless food after a time leads to distaste of food; so that the addition of flavouring agents is an absolute necessity in physiological conditions of life.

Food accessories may be divided into two classes, condiments, and a class containing alcoholic beverages, tea, coffee, and cocoa. Both classes of substances possess somewhat similar physiological actions, which are mainly of two kinds, an action on the process of digestion in the stomach, and an action on the central nervous system.

The chief condiments in use are mustard, pepper, onions, garlic, pimento, cloves, cinnamon, nutmeg, caraway, cardamoms, and vinegar, to which list common salt must be added. The active principles of these substances (with the exception of vinegar and common salt) have a threefold action.

1. They are *antiseptic;* especially oil of mustard, onions, pimento, cloves, and the other aromatics.

2. They *stimulate the secretion of the digestive juices.* They without doubt stimulate the secretion of the saliva, and thus indirectly the secretion of the gastric juice; but it is possible that they also stimulate directly the secretion of gastric juice when they enter the stomach.

3. They *stimulate peristalsis.* Some of the active principles (especially the aromatic oils) also act as stimulants to the nervous system, but their effect is not great in the small doses in which they are taken as condiments.

Besides their pleasant smell and taste, and appetising effect, these condiments are of distinct value in healthy digestion.

The second class of food accessories is a large one, and may be subdivided into liquids containing alcohol and those containing alkaloids (such as tea, coffee, and cocoa).

Alcohol and the alkaloids, caffeine, and theine, have a special action on the central nervous system, being in small doses stimulant and in larger sedative. It is probable that such drinks are largely taken for this object as well as for their pleasant aromatic taste.

They act, moreover, and this is especially the case with alcoholic beverages, as stimulants of the secretion of gastric juice, which in part explains their action when taken with meals in some cases of disordered digestion.

Lastly, they act directly on the chemical processes of digestion in the stomach.

In artificial digestion experiments with pepsin-hydrochloric acid, Sir William Roberts[1] obtained the following results:—

Proof spirit present in the digestion-mixture to the extent of 10 to 20 per cent retarded the chemical processes. The ardent spirits (brandy, whisky, and gin) retarded the digestion in proportion to the amount of alcohol they contained; and thus in dietetic doses would not have any effect on the chemical processes of digestion in the stomach. It is quite otherwise with wines and beers: these retard digestion out of all proportion to the alcohol they contain; tea, coffee (especially *café noir*), and beef-tea having a similar effect. The alcoholic beverages which have the most energetic action in this respect are sherry, port, and beer; 20 per cent of sherry, for example (equal to 8 per cent of proof spirit), trebling the time of digestion. Hock, claret, and champagne are less active: champagne having the least retarding effect, probably from its effervescence.

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[1] "Dietetics and Dyspepsia" (Lectures at Owens College, 1885). See also Schellhaas, *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxxvi. p. 427; Schütz, *Prag. med. Wchnschr.*, 1885, No. 20; Büchner, *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxix. p. 537. All these researches gave practically similar results to those of Roberts.

It is noticeable that tea and coffee have no retarding effect on pancreatic digestion, and wines and beer have an effect only in proportion to their degree of acidity.

It is highly probable that part of the retarding effects of wines and beer on the chemical processes of digestion is due to the presence in them of organic salts (lactates, tartrates, etc.), which in the presence of hydrochloric acid are decomposed, the free acids being liberated. In the presence of these acids digestion by pepsin-hydrochloric acid is retarded. Part of the slowing action may be due to the inorganic salts present, chiefly those of potassium. Sodium chloride up to 1 per cent increases the chemical processes of digestion.

The effects of alcohol and alcoholic drinks on the chemical processes of digestion outside the body which have just been described are of great interest, but do not accurately indicate the occurrence of events in the stomach itself during actual digestion. It is commonly said that small doses of alcohol before meals promote digestion by increasing the secretion of gastric juice; this has been confirmed by experiment, although for many reasons it is not a custom to be advocated, except in rare instances.

An early observation of Claude Bernard[1] showed that the difference between the action of alcohol and of ether on digestion was that the former prevented while the latter increased digestion. Thus two dogs which had the same meal were given, the one ether and the other alcohol, in anæsthetic doses; both were killed in five to six hours, and digestion in the etherised animal was far advanced or ended, while in the alcoholised animal it had barely begun. Kretschky[2] made some observations on a young girl with a gastric fistula and found that a small dose of alcohol (3 per cent of absolute alcohol) delayed digestion when given with the meal, and in another case of fistula Richet[3] found that alcohol disappeared from the stomach in thirty-five to forty minutes, and that not only wine but pure alcohol increased the acidity of the gastric juice. Thus, although when wine is given part of the increased acidity is due to the acids it contains, yet this cannot be the explanation of the increased acidity when pure alcohol is given. As Gluzinski's[1] observations show, alcohol actually increases the secretion of hydrochloric acid by the stomach. The method he used was as accurate as such methods can be. Individuals who were healthy and those who had disease of the stomach, those who were accustomed to alcohol and those unaccustomed to its use were examined. When the stomach was empty, the patient was given the white of one egg coagulated, and 100 cubic centimetres of distilled water. The patient remained quiet for a certain period, after which 100 cc. of water were injected into the stomach, and the contents removed by an aspirator.[2]

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[1] "Leçons sur les effets des substances toxiques et médicamentaires." Paris, 1875, p. 430.

[2] *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xviii. p. 527.

[3] "Recherches sur l'acidité du suc gastrique chez l'homme," etc.—*Compt. rend. Acad. d. Sc.*, Paris, tome xxxiv., 1877.

With this simple food, therefore, a very accurate idea could be obtained of the process of digestion in the period after giving the egg-albumin. The ordinary time of digestion of this meal was investigated in each individual, when no alcohol was given; afterwards an exactly similar experiment was performed, with the exception that instead of giving water with the egg-albumin, 100 cc. of 25, 50, or 75 per cent alcohol was substituted; or a small glass of cognac. The fluid which was withdrawn from the stomach after digestion was tested for the degree of acidity, for the presence of alcohol, and for the degree of digestion of the pieces of egg-albumin, and for the digestive activity of the fluid, *i.e.* as regards the amount of pepsin it contained. Three individuals with normal digestions were investigated: one was suffering from sciatica, the second from hysteria, the third was convalescent from malaria. The first patient only was accustomed to take alcohol, and that in moderate quantities. Without going into too great detail it may be said that the results obtained were as follows:—Alcohol soon disappears from the normal stomach, its disappearance being most rapid with a small dose, and when there is only a small quantity of food in the stomach. The alcohol in 100 cc. of a 25 per cent mixture is not present in the stomach half an hour after taking it: that in 100 cc. of a 50 per cent disappears in three-quarters of an hour, and that in 100 cc. of a 75 per cent mixture in one hour.[1] The most important conclusions of Gluzinski's experiments deal with the effect of alcohol on the digestion of proteids by the gastric juice. The process of digestion in the stomach when alcohol is taken with the food may be divided into two stages.

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[1] *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxxix. p. 405, 1886.

[2] See Chapter V., Methods of Examination of the Stomach.

1*st Stage.* There is an inhibition or prolongation of digestion as compared with the period of digestion in the same individual when alcohol is not taken. In this stage alcohol is still found in the stomach, and the duration of the stage depends on the dose given. Then in the experiment with 100 cc. of 25 or 50 per cent alcohol, the duration was fifteen to thirty minutes; with a similar quantity of 75 per cent alcohol it was forty-five minutes. The secretion of hydrochloric acid is not effected in this stage, and the prolongation of digestion may be due to the precipitation of the pepsin by the alcohol present.

2*nd Stage.* When the alcohol is absorbed by the stomach, the second stage begins, in which there is increased digestion, so that with moderate doses of alcohol the digestion of the meal given may be completed in about the same time as when none is taken. This increased activity of digestion appears to depend on a greatly increased secretion of hydrochloric acid, the acidity of the gastric contents rapidly increasing in this stage. Moreover, the secretion of gastric juice goes on for some time after digestion is ended, thus showing that alcohol in this second stage actually stimulates the gastric glands to secrete. The mechanical powers of the stomach are but slightly influenced by alcohol in moderate doses: it is only when large doses are given that the movements are deficient.

Although the digestion of albumin is the most important process that occurs in the stomach, yet when taken in such small quantity as in Gluzinski's experiments and with relatively so large a quantity of alcohol, the process of digestion cannot be considered as exactly comparable to that of a moderately large mixed meal. In one individual the digestion of a mixed meal was tested. A comparison was made between the digestion without alcohol and that with alcohol. The meal in each experiment consisted of soup, a beef-steak weighing about 5 ounces (140 grammes), and a piece of bread. The stomach contents were removed and examined in three hours, four hours, and five hours. In the digestion of three hours' duration it was found that in the absence of alcohol, the process had progressed more rapidly than when alcohol was given; but that in four or five hours there was no difference in the amount of digestion with or without alcohol. The amount of alcohol given was 100 cc. of a 50 per cent solution. Gluzinski's observations deal with the action of alcohol on digestion in the stomach; they, however, leave untouched the action of alcoholic drinks, all of which, except the ardent spirits, retard very greatly the chemical processes of digestion, as the experiments of Roberts, Büchner, and others show.

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[1] It may be mentioned that Gluzinski did not find any aldehyde in the stomach, and thus could not confirm Kretschky that this body was formed from alcohol in that organ. Alcohol appears to enter the circulation as such.

As a summary of this part of the subject it may be said that the proper performance of the functions of the stomach depend on the following conditions:—

1. As to the stomach itself, there must be a regular and sufficient secretion of gastric juice during digestion; the movements of the organs must be also continuous and regular, so that the food should be intimately mixed during digestion and not too soon propelled into the duodenum.

2. As to the food taken:

(*a*) In any quantity, it must be taken at regular intervals of not less than four to five hours.

(*b*) The food at any meal must be properly prepared and cooked.

(*c*) The food must not be too acid, and not of too great bulk at one meal.

(*d*) The food must consist of a certain proportion of proteids, carbohydrates, fats, salts, and water: a preponderance of one or other constituent of the diet leading to disturbances both general and local.

(*e*) The food accessories must not be in too great amount, since an excess, especially of alcoholic beverages and tea, etc., leads to digestive disturbance.

CHAPTER III.

PATHOLOGY OF INDIGESTION OF FOOD.

On the regular observance of the dietetic rules, which have been discussed in the preceding pages, normal digestion, which is easy and painless, depends: a continuous breach of the rules leads to disordered digestion. Disordered digestion, or indigestion of food, does not, however, always depend on the breach of dietetic rules. This leads to alterations in the secretion, movements, and absorption of the organ; but a similar change may be due to a general disease of the body, or to organic changes in the stomach itself. In both *functional* and *organic* diseases of the organ, therefore, indigestion of food occurs; and it is important to study the causes of this indigestion, in order to clearly distinguish the effects of organic disease from those of functional disease of the organ.

From a pathological point of view, indigestion of food may be tabulated under the following heads:—

A.—Local Causes in the Stomach.

1. Due to the character of the food itself.
    - *a.* Its degree of subdivision when received into the stomach.
    - *b.* Its bulk, composition, and chemical reaction.
    - *c.* The amount of food accessories taken.
    - *d.* Drugs and salts taken with it.
2. Due to fermentative changes in the food, associated with delay of food in the stomach.

3. Due to changes in the functions of the stomach.
    a. Organic disease being absent.
    b. Organic disease of the organ being present: inflammation, degeneration, ulcer, or new growth.
        a. Affecting the secretion of gastric juice.
        b. Affecting the movements of the stomach.
        c. Affecting the absorption.

Changes in the functions of the stomach when organic disease of the organ is absent may be due either to the food itself or to a disease of the body other than that of the stomach. These general causes may be tabulated as follows:—

B.—General Causes.

1. General and local non-infective disease affecting the functions of the stomach: such as chlorosis, heart disease, Bright's disease, liver disease, gout, and functional and organic diseases of the nervous system.

2. Infective disorders affecting the functions of the stomach: such as tuberculosis, malaria, typhoid fever, scarlet fever, measles, cholera, rheumatic fever, influenza.

Local Causes in the Stomach.

Causes of Indigestion to be ascribed to the Food taken.—The food taken may be a cause of its own indigestion, but in the majority of cases it is the direct cause of the symptoms and the means by which the disease is aggravated.

1. The first essential for good digestion in the stomach is efficient mastication; first, in order that some of the starch should enter the stomach in a partially digested condition, and thus that there should not be in the stomach a large quantity of material which the gastric juice is unable to digest, dissolve, or manipulate; and, secondly and chiefly, in order that the food should enter the organ in as finely divided a state as possible. If the food is not well chewed, it enters the stomach in lumps, on which not only does the gastric juice act very slowly, but which the stomach finds extremely difficult to propel through the pylorus. The effect of subdivision of food as an aid

to the chemical processes of digestion is easily seen in artificial digestion in which a mixture of pepsin and hydrochloric acid will readily digest a solution of egg-albumin which is coagulated in fine flocculi, but will act in the same time to a very slight extent on a piece of the white of a hard-boiled egg.

Imperfect subdivision of food may be due to the following causes :—

(1) To the toughness of the food.
(2) To the habit of rapid eating and bolting of the food.
(3) To pain caused by decayed teeth.
(4) To the absence of teeth.

The first three points need no commentary. As regards the fourth, it is important to remember that mastication of the food is performed by the molar teeth, and people who have lost the majority of the molar teeth use the front teeth (and very inefficiently) for mastication. It is necessary that there should be some teeth opposite to each other in each jaw.

In some cases, imperfect mastication of the food is the sole cause of disordered digestion; in very many, it is one of the causes. The manner in which it disorders digestion is that it causes delay of food in the stomach, owing to the gastric juice being unable to reduce to a pulp the masses of food, which are either vomited or discharged into the duodenum with great difficulty and with pain.

2. The *bulk, composition, and reaction* of the food are the next points to be considered in the production of disordered digestion (Chapter II.).

As regards bulk, the normal stomach is capable of holding about 35 to 40 ounces of liquid. It has been stated that the stomach can at any one time manipulate a large quantity of food received into it without any distress on the part of the person. Disorders, however, arise when a large quantity of food is regularly taken into the stomach at each meal: a large breakfast, a large lunch, and a larger dinner. The amount of food habitually eaten by many people in this way daily is very great.

The daily amount of food (weighed dry) which is necessary for an adult man doing moderate work has already been stated to be about 65 ounces, to which must be added about 70 ounces of water. This weight of food is frequently exceeded in the well-to-do, and leads to disordered digestion *from its mere bulk* in two ways.

In the first place, a large amount of work is thrown upon the stomach in manipulating a large meal; in the second place, if large meals are frequently repeated, they disturb digestion not only by the fact that one meal is not nearly digested by the time the second is taken, and thus the stomach is never empty, but by the fact also that the stomach is constantly at work, and although the first stage may be one of hypersecretion, its functions, both as regards the secretion of gastric juice and the movements, finally diminish in activity. Large meals therefore end in producing non-digestion of food, and especially so when taken two or three hours before going to bed, as during sleep digestion practically ceases. It is quite true that many people can for years continue to consume large quantities of food at each meal without any apparent harm; but such excess, which is apparently harmless in young adults, becomes a habit in middle age, and it is at this period, critical as it is for many diseases, that the effects of excessive eating are most apparent.

In excessive eating, the bulk of the food is only one of the factors producing disordered digestion; the character of the food and the quantity of food accessories taken also play an important part.

As regards the composition of the food, the laws regulating the diet which will maintain health have already been discussed (p. 40).

The fault in the dietary of the well-to-do is that there is a tendency to take too much proteid food; in other words, to excessive meat-eating. In the dietary of the poorer classes, meat being an expensive article of diet, an excess of carbohydrates is taken. Excessive meat-eating has a twofold effect, one directly on the stomach and one on the metabolism of the body, or, in other words, on the nutritive changes occurring in the tissues. A large quantity of meat eaten regularly each day gives the stomach too much work to do, since the action of the gastric juice on the meat is a necessity for its digestion which is completed by the pancreatic juice. If the practice is continued, like all overworked organs, the stomach ceases to secrete the proper amount of juice, and ceases to have active movements; the result is non-digestion of food.

The effect of excessive meat-eating on the metabolism of the body has already been alluded to. If there is an excess of proteid food stuff in the diet, there is usually a diminution in the amount of fat and carbohydrates taken. Thus with a diet in which meat preponderates largely, the body draws on its store of fat and consequently wastes; a large amount of work is thrown on the tissues by which the absorbed proteid is utilised and excreted as urea.

Carbohydrates and fats differ from proteid foods, such as meat, in the fact that they undergo no chemical change in the stomach; pepsin-hydrochloric acid having no action on them. Fats are freed from their enveloping proteid material in the stomach, and are the last part of the food to leave the organ. When a large quantity of fat is taken it leaves the organ with difficulty or not at all, remaining there to undergo decomposition. The actual presence of a large quantity of fat also delays digestion in the stomach by interfering with the fine subdivision of the food which occurs under normal conditions.

With regard to carbohydrates, and especially starch, it is of great importance to digestion in the stomach as to in what condition they arrive in the organ. A partial digestion of the starch by the saliva in the mouth, transforming it into dextrin and maltose, aids digestion in the stomach since the products are soluble, and are soon absorbed or pass through the pylorus. If, however, the starch or a large quantity of it is unaffected by the saliva, it enters the stomach in an insoluble form, and one which mechanically interferes with the chemical processes of digestion by the gastric juice. In many people the saliva is but little active, and this effect is noticeable. The important effect of an excess of one or other constituent of the dietary is that thereby digestion is delayed, and food remains unnecessarily long in the stomach and, as will be seen, may undergo fermentative changes.

Besides the effect of an excess of carbohydrates in the diet, which has just been referred to, there are others which are exciting causes of the indigestion of food. The more carbohydrates that are eaten, the more indigestible cellulose is taken, as this is a constituent in all vegetable food, and in some is very abundant: also with an excess of vegetable food, an excess of organic acids and salts is taken, which increase the acidity of the stomach contents, but delay digestion, since gastric digestion is less rapid in the presence of these acids than in that of hydrochloric acid, and lactic, acetic, citric, and tartaric acids must be considered as inimical to active digestion in the stomach.

An excess of cellulose in the diet not only delays digestion in the stomach, but diminishes the total digestion and absorption of the food, more nitrogen being present in the fæces under such conditions than when the diet is a normal one (p. 47).

The amount of cellulose present in the commonly used vegetable foods is a point worthy of further consideration, as it is one of the causes of the indigestion of vegetable foods.

The most digestible vegetable foods contain the least cellulose: these are wheaten, barley, and rice flours, potatoes, spinach, and cauliflower. Fine wheaten flour contains as little as 0·29 per cent of cellulose, while coarse flour ("seconds") will contain nearly 1 per cent, and whole meal flour 1·9 per cent. Barley and rice flours each contain nearly 0·5 per cent; potatoes 0·69 per cent, spinach and cauliflower nearly 1 per cent. Other cereal flours (such as oat, rye, maize) and other vegetables contain over 1 per cent of cellulose, fine oatmeal containing as much as 1·86 per cent. From the point of view of the amount of cellulose present, as well as from other points of view, wheaten flour is the most digestible form in which carbohydrates can be administered in the form of starch. This is particularly so, since it can readily be prepared in a palatable form. In the case of baked flour, some of the starch is transformed into dextrin, a transformation which is distinctly a gain to the proper performance of digestion in the stomach; in the form of bread, flour is really a partially digested food, since in the process of fermentation and baking through which the flour goes, a good deal of the carbohydrates is transformed into dextrin and sugar, and some of the gluten is broken up into more soluble proteid bodies. Bread is only indigestible when it is not porous, when it contains too much water, or too much cellulose (as in brown and whole-meal bread).

Of the other carbohydrates taken as food, cane-sugar is the most likely to produce indigestion of food; maltose, lactose, and dextrose to a less extent.

Regarding the chemical reaction of food, it need only be said that food which is too frequently taken in an acid condition leads to delay of digestion. A certain amount of acid (*e.g.* vinegar) is a necessity with some foods, *e.g.* with vegetables, partly as a flavouring agent, and partly for the needs of the organism; so too the acid fruits act beneficially in a dietary. But an excess of these vegetable acids leads to derangement, as in a case in which a man acquired the habit of taking citric acid in water with meals and in between, for a long period (over twelve months), with the result of producing indigestion of food and the passage of alkaline urine, the symptoms ceasing on discontinuing the practice.

The importance of cooking in ensuring the proper digestion of food in the stomach cannot be over-estimated. Food imperfectly prepared may have two effects. The state in which meats are most easily digested is when the proteid constituents (myosin, etc.) are not completely coagulated, so that the gastric juice can readily act on them; if too much cooked, or twice cooked, they become completely coagulated and form a hard mass, on which the gastric juice acts slowly. The over-cooking of meats therefore leads to delay of food in the stomach. The cooking and preparing of certain foods may also result in a pasty or hard mass, which it is difficult for the stomach to manipulate; as, for example, in imperfectly cooked pastry and in doughy bread.

Food Accessories as a Cause of Indigestion of Food.—The physiological effects of food accessories have already been considered (p. 49). It is now necessary to deal with the effect they have in producing indigestion of food in the stomach.

The essential oils and other active principles existing in condiments are beneficial to digestion in the stomach up to a certain point; they are, however, habitually taken not for any effect they may have in this way, but solely as flavouring agents in the food. In excess they act as irritants (p. 69). Some condiments, such as pickles, which are mixed with a large amount of vinegar (acetic acid), have a particularly deleterious effect on digestion when taken in excess; and this is not only due to the acid or other irritants they contain, but to the use of vegetables containing a large amount of cellulose.

Turning now to the other food accessories, viz. alcoholic drinks, and tea, and coffee; it has been shown that these have a retarding effect on the chemical processes of digestion (p. 49) in the stomach, and that this result is not proportional to the amount of alcohol they contain, but is due to certain other constituents: organic bodies and inorganic salts. Thus the ardent spirits (brandy, whisky, etc.) have in dietetic doses but little effect on digestion in the stomach, while port and sherry are very harmful in this respect, and beer, hock, and claret harmful, but to a less extent than port and sherry. Tea and coffee act in a similar way. The retardation of the chemical processes of digestion means a delay of the food in the stomach and its attendant changes, but it has already been pointed out that alcoholic drinks contain certain organic salts (lactates, malates, tartrates, etc.) which in the stomach are decomposed, the acid component being set free. In the presence of this increased acidity due to organic acids, gastric digestion does not proceed so well as when hydrochloric acid is in preponderance. As regards the amount of these organic salts present in alcoholic drinks, it may be said that the percentage is not quite proportional to their retarding effect on the chemical processes of digestion. Brandy and whisky contain only a trace of organic acid (acetic); port and sherry about 0·40 to 0·45 per cent reckoned as tartaric acid; English beer contains about 0·28 per cent of lactic acid; French red wine about 0·6 per cent of the same acid, white wine and champagne containing rather more.

The delay of food in the stomach caused by food accessories may therefore be not only due to a retardation of the chemical processes, but may lead to increased acidity of the stomach's contents, an acidity due to irritating organic acids.

These effects will be the more marked, the larger the quantity of alcoholic drink or tea and coffee taken with food, and will be very manifest too if such drinks are taken in excess between meals when digestion is at its height.

In the preceding paragraphs stress has been laid on the fact that the non-observance of certain dietetic rules leads to indigestion of food in the stomach and to one particular result, viz. *delay of food in the stomach.* Imperfect subdivision of the food, imperfect cooking, too great bulk of the food, too great a preponderance of one or other constituent of the dietary, an excessive amount of food accessories, and too great an acidity of the food may lead to delay of food in the stomach and to acidity of the stomach contents due to organic acids, a kind of acidity which delays the chemical processes of digestion. Delay of food in the stomach is the cardinal fact, and leads to changes in the food, chiefly fermentative, and to effects on the stomach, which will be the subject of frequent consideration later. But the period of stay of food in the stomach may be due not only to the conditions of the food which have been just considered, but to changes in the functions of the stomach itself, whereby the secretory activity, the movements, and the absorptive powers of the organ are affected.

The causes of delay of food in the stomach may be summarised as follows :—

A.—*On the Part of the Food Itself.*

1. Imperfect subdivision of the food.

2. Over-cooking or imperfect cooking.

3. Too great a bulk of food at one meal.

Thus delaying digestion by giving the stomach too much work to do, or too large lumps of solid food to manipulate.

4. Too great a preponderance of one or other food stuffs: of meat, which entails extra work for the gastric juice: of fats and carbohydrates, which are unaffected in the stomach, and, in excess, are difficult to expel from the organ.

5. Too large an amount of indigestible matter, *e.g.* cellulose mixed with the food.

6. An excessive amount of food accessories, taken with or after meals; delaying the chemical processes of gastric digestion.

7. Excessive amount of vegetable acids or salts, *e.g.* lactic, acetic, tartaric acid and their salts; these acids are inimical to gastric digestion, which is less vigorous in presence of these than in that of hydrochloric acid.

B.—*On the Part of the Stomach Itself.*

1. Alterations in its functions, from many causes, organic or temporary, which produce :—

(*a*) Diminution in the amount of pepsin and hydrochloric acid secreted, whereby the chemical processes are delayed.

(*b*) Diminution in the movements of the stomach, whereby the food is not churned and not propelled normally through the pylorus.

(*c*) Diminution in the absorption in the stomach.

2. Alteration in its structure :—

(*a*) The presence of a growth or ulcer in any part causing deficient movement or defective secretion.

(*b*) Obstruction of the pylorus by a new growth, cicatrising ulcer or fibrous contraction, or by pressure or traction from without, whereby there is difficulty of expulsion of the contents of the stomach.

The results of delay of food in the stomach are of great importance in the study of diseases of the organ, both functional and organic. During efficient digestion, *i.e.* digestion without disturbance, the stomach empties itself in a certain time, which for a large meal may be reckoned as from five to seven hours, and for a small meal as about three hours. At other times, the stomach is resting, there are no spontaneous movements of the organ and no secretion of gastric juice, the contents being a small amount of alkaline liquid with a few flakes of mucus.

If the period of stay of the food is delayed from any of the causes enumerated above, an amount of work is thrown on the stomach which it is unable to perform, and the difficulty is surmounted by ejecting the food through the mouth or passing it in an undigested form through the pylorus, a result in either case obtained after great distress.

The question is, however, not simply one of a large amount of work thrown on the organ, but the condition is aggravated by the fact (1) that the organ is always at work, and never empty, one meal being taken before the other is disposed of; and (2) that the continued presence of food in the stomach acts in the course of time as an irritant, producing congestion and an excessive secretion of mucus, and frequently an excessive secretion of hydrochloric acid, and also in the end acts in an inhibitory manner on all the functions of the organ.

An apt illustration may be taken in the case of a person who, healthy, but unaccustomed to large meals, takes a very large meal, *e.g.* at a public dinner. In this case, there are no obvious results during the two hours or more in which the meal is being consumed, a meal, it must be remembered, consisting of a large amount of meat, flavoured in various ways, a large amount of liquid, and a large amount of alcoholic drinks. With such a meal, the chemical and mechanical processes of digestion progress for a time normally; *i.e.* the gastric juice secreted acts on the proteid constituents of the meal, and assisted by the mechanical movements reduces the food to the proper consistence of chyme. But with the progress of the meal, and after it is concluded, certain events are occurring which interfere with the digestion of the food and cause its delay in the stomach. Thus the bulk of food taken into the organ becomes greater and greater, and exceeds the amount which the organ can manipulate: this is one cause of delay. Next, both with the food and with alcoholic drinks is taken in the course of so large a meal a large quantity of vegetable acids and salts, which being all in the condition of free acids in the stomach, render the contents of the organ very acid, but with an acidity due to organic acids which are not so favourable to gastric digestion as hydrochloric acid: this is the second cause of delay. Then the large amount of alcohol taken also delays the chemical processes of digestion: this is the third cause of delay. After the meal, therefore, all these three causes acting, the normal processes of digestion, both chemical and mechanical, diminish and finally cease. The result is that there is remaining in the stomach a grumous liquid, very acid, which the organ is incapable of propelling through the pylorus. In many instances it is therefore vomited in six to ten hours with relief of all the symptoms experienced. These events occur in individuals who are healthy but unaccustomed to take a large meal with alcoholic drinks. It does not always occur, and that it does not is dependent on the fact that the healthy stomach is capable of doing a large amount of work at one time.

If large meals are repeatedly taken, although the stomach may become more or less accustomed to them, in the majority of cases disordered digestion results. The symptoms observed are those usually ascribed to "acid dyspepsia" (gastric irritation), in which there are acid eructations and discomfort occurring in one to two hours after food. Fermentation of the food by means of bacteria is not an accompaniment of this condition. Organic acids derived from the food itself are present in the stomach contents, and in conjunction with an excessive secretion of hydrochloric acid, which frequently occurs, cause irritation of the mucous membrane of the overworked stomach with the consequent acid eructations. In this form of "acid dyspepsia," therefore, the acid which irritates is partly the hydrochloric acid secreted in the gastric juice, and partly the organic acids (lactic, acetic, butyric, etc.) taken with the food and the food accessories (chiefly wines and beer). The frequent repetition of large meals in the day, and day by day, leads to more changes than these just mentioned. The delay of acid food in the stomach is one result, but the occurrence of this for a long period leads to a diminution in the normal functions of the stomach; in a diminution of the amount of gastric juice secreted, in a diminution of the normal movements of the organ, and in a diminution in the amount of absorption.

Up to a certain point, additional work placed on any organ of the body leads to increased work of that organ, a familiar example being the hypertrophy of muscle during a course of training for athletic purposes. A continuance of additional work in time, however, leads to a diminution of the functions of an organ; an over-trained athlete cannot get a proper amount of work out of his muscles. As with a muscle, so with the stomach, which is more constantly overloaded with work than any other organ of the body. The frequent repetition of large meals leads in the first instance to increased work of the organ, frequently associated with hypersecretion of hydrochloric acid, but eventually it leads to a diminution in the secretion of gastric juice and to a diminution in the movements of the stomach. Thus the delay of food in the stomach, which is produced by the repetition of large meals, is increased, since by the diminution of the gastric juice the chemical processes of digestion are delayed, and by the diminution of the movements the expulsion of the semi-digested food (chyme) through the pylorus into the duodenum is diminished. In these prolonged cases of functional disturbance, the delay of food in the stomach leads to other changes in the stomach contents, viz. bacterial fermentation of the food. Another important change also occurs which further aggravates the condition, viz. dilatation of the stomach. The organ never being quite empty, and having its movements diminished by the various causes mentioned, and the contents undergoing fermentation, which produces gas distending it, it dilates; becoming so enfeebled that it is never in the normal contracted state between meals (Chapters IV. and XIV.).

The effect, therefore, of the continued repetition of large meals is a delay of food in the stomach, and its consequences, viz. (1) the constant presence of an acid semi-liquid which acts as an irritant; (2) the subsequent diminution in the normal functions of the stomach as regards the secretion of gastric juice and the movements; (3) the bacterial fermentation of food in the stomach; and (4) the dilatation of the organ.

The amount of food that can be eaten with impunity by healthy individuals varies according to the build of body, to the age and sex, to the mode of life whether one of active exercise or chiefly sedentary, and to the condition of the nervous system. Healthy individuals are also susceptible to certain articles of diet, which if eaten lead to delay of food in the stomach and its results. Beyond a certain amount of food all individuals, however, suffer, and especially is this so between the ages of forty and fifty, a critical period for many previously healthy individuals.

It is from the point of view of *food acting as an irritant* to the stomach that dietetics becomes of such great importance in the treatment of disordered digestion. As has been just discussed, the mere bulk of the food may act as an irritant as well as the presence of a large amount of indigestible matter (*e.g.* cellulose), or of food-stuffs which cannot be easily digested by the stomach at all (*e.g.* starch and fat). An increased organic acidity caused by the food will also act as an irritant, either when there are free organic acids taken with the food (*e.g.* acetic or citric acid), or when there are salts of these organic acids present, for they are decomposed by the acid gastric juice. Ordinary simple articles of diet do not contain organic acids or salts in amount sufficient to irritate the normal stomach, when they are eaten as fresh food; when smoked or otherwise prepared they may, however, be too acid. Thus meat in the fresh condition contains from 0·05 to 0·07 per cent of sarcolactic acid, but when smoked it may contain as much as 0·72 gramme per cent. Similarly fresh pork, containing 0·36 gramme of lactic acid in every 100 grammes, may contain 0·72 to 0·855 gramme per cent when made into sausages.[1] Butter (fresh) contains only a trace of free acids (0·005 to 0·01 per kilo., Duclaux), and is a non-irritating fat; buttermilk may contain from 0·11 to 0·62 gramme per cent of lactic acid, or on an average 0·34 per cent (König). Similarly with vegetable foods, the percentage of organic acids varies greatly. Wheaten flour, *e.g.* contains practically no organic acids, while bread contains a fair proportion, which increases as the bread is kept (p. 33). The organic acids (lactic, butyric, and acetic) are the products of the fermentation of the carbohydrates during the process of manufacture. Green vegetables, and the starchy vegetables as well as fruits contain malic, citric, and tartaric acids, which all increase the organic acidity of the stomach; while some fruits contain oxalic acid, which is more irritating than the other acids. With regard to the effect of these organic acids in causing irritation of the stomach, it is not so much a question of the quantity taken at each particular meal, but of

[1] Kämerer, quoted by König, *op. cit.* Bd. ii. p. 168.

the continued use of food containing those acids or their salts, or highly flavoured with acid sauces. When a condition of irritation of the stomach is established, these acids greatly aggravate the condition.

Something has already been said about the physical condition of the food as regards its digestibility; hard food and dried food (dried meat, fish, etc.) act frequently as irritants, since their stay in the stomach is a very prolonged one.

Of greater effect as a means of irritating the stomach is over-indulgence in food accessories, viz. alcoholic drinks and tea. The effect of alcoholic drinks has been shown to be a delay in the chemical processes of digestion, and in this way by causing the food to remain a long time in the organ they act as irritants. Their irritant result is, however, not solely nor chiefly, due to this effect. Alcohol is itself a local irritant, producing dilatation of blood vessels, and thus its long continued use leads to irritation of the stomach and a chronic catarrh. Moreover, its physiological action on gastric digestion has been shown by Gluzinski (see p. 52) to be divided into two periods, in the first of which digestion is delayed and in the second increased. This increase of digestion is due to a hypersecretion of gastric juice (and especially of the hydrochloric acid) which may continue after digestion is completed. The stomach mucous membrane is therefore exposed to the irritating effects of this hyperacid gastric juice when food is absent. Alcoholic drinks lead to hyperacidity of the gastric juice also in another way; they contain tartrates and malates, which are decomposed by the gastric acid forming free organic acids. Not only therefore by its causing a delay of food in the stomach, but by its local action on the mucous membrane, and mainly by its effect in causing a hyperacidity of the gastric contents, alcohol as well as alcoholic beverages acts as an irritant to the mucous membrane, and leads to chronic catarrh.

Of the other food accessories, tea is a very common cause of irritation, especially the cheap kinds which contain a large quantity of tannin, and are infused for long periods. Spices, curry, etc., all act as local irritants to the mucous membrane, but this effect is minimised if taken in small quantity with a large meal.

2. *Bacterial Fermentation of the Food in the Stomach.*—Bacteria are constantly being taken into the stomach with the food, but their development is hindered chiefly by the presence of hydrochloric acid in the gastric juice. This acid, when free, is inimical to the growth of micro-organisms; but when loosely combined with proteids, its effect in this respect is much diminished.[1] As Beaumont showed in the case of Alexis St. Martin, the gastric juice when removed from the stomach can be kept for a long period without undergoing putrefaction or becoming altered in its properties, and more than a century ago Spallanzani[2] demonstrated the antiseptic action of the gastric juice. Some observers, indeed, consider that the chief *rôle* of the gastric juice is that of preventing the entrance of micro-organisms into the alimentary tract. Bunge[3] has expressed a very decided opinion from this point of view, and considers that the chief action of the hydrochloric acid is an antiseptic one. This is an exaggerated view to hold, and cannot be maintained in face of the facts relating to the fermentative activity of the pepsin-hydrochloric acid, which have already been described (Chapter I.). Although it is probable that the pancreatic juice is the chief agent in digestion, yet the action of the gastric juice is essential, as we have seen, not only in preparing the mixed food for intestinal digestion, but for reducing the proteids to a condition in which the trypsin of the pancreatic juice can readily and efficiently act upon them.

Bacteria are taken into the stomach chiefly with uncooked food and unboiled liquids. If fresh food, properly cooked, is eaten hot, very few micro-organisms are swallowed. On the other hand, with food, especially meat and green vegetables, which have been over-cooked and preserved (either tinned or otherwise), bacteria are in some cases present, and some of the most virulent cases of bacterial gastro-intestinal infection occur in this way.

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[1] Hamburger, *Abstract in Centralbl. f. d. med. Wissensch*, Berlin 1890, p. 726.

[2] "Dissert. relative to the Nat. History of Animals and Vegetables," vol. i., Dissert. 6, p. 251, London, 1784.

[3] Bunge, "Lehrbuch der physiolog. und patholog. Chemie," 3rd ed., 1894, p. 140 *et seq.*

It is known that the acid contents of the healthy stomach do not kill the majority of the bacteria taken with the food, although their development is hindered, and it has been proved by direct experiment that they can pass in a living state through the stomach into the intestines (*e.g.* staphylococcus aureus, micrococcus tetragenus) (Macfadyen); and the spores of bacteria more readily pass than the developed forms. Large numbers of bacteria do pass through the stomach unharmed, since they are found in abundance in the intestine, and may be obtained by cultivation from the intestinal contents.

Although much work remains to be done in the way of distinguishing the bacteria of the stomach and intestine, and in the study of their life-history and injurious effects, yet it is sufficient to add here that some of these bacteria under certain circumstances enter the mucous membrane, and produce an infection of the stomach and intestines (Chapter IX.), while others affect the stomach and intestinal contents decomposing them, and producing bodies which are harmful. It is the effect of the latter class of micro-organisms that will now be considered.

In a bacteriological investigation of the stomach contents in thirty individuals, Capitan and Moreau[1] found only three kinds of micro-organisms, two of which were moulds and one a bacillus. Abelous[2] in an investigation on himself found sixteen different forms in the stomach washings: seven of them were known, viz. sarcina ventriculi, bacillus pyocyaneus, bacterium lactis aerogenes (Escherich), bacillus subtilis, bacillus mycoides, bacillus amylobacter, vibrio rugula—of the remaining forms, eight were bacilli, and one was a coccus. These micro-organisms were found able to withstand the action of hydrochloric acid (0·17 per cent) for a long time, especially if they contained spores. Four of them peptonised the casein without coagulating it, nine coagulated the milk and dissolved the coagulum, and four coagulated the milk without dissolving the clot. Carbohydrates (lactose, cane-sugar, glucose, and starch) were more or less affected by the micro-organisms.

Owing to the presence of hydrochloric acid in the stomach contents, the bacteria taken in with the food do not develop during normal digestion; but when there is a deficiency of the hydrochloric acid, then they can develop and produce their harmful products. A certain proportion of hydrochloric acid undoubtedly acts as an antiseptic, preventing the development of micro-organisms, but the resistance of individual micro-organisms to the action of hydrochloric acid varies. As regards the pathogenic forms, the cholera vibrio is readily killed, bacillus anthracis not so readily, and bacillus tuberculosis still less readily. The acid-forming non-pathogenic micro-organisms are said to be more resistant than the pathogenic, *i.e.* their action is inhibited, but they are not killed by the hydrochloric acid. Rummo and Ferannini[1] found that 0·05 per cent of the acid delayed the alcoholic, the lactic and butyric acid fermentations; the effect being much more evident if the percentage of acid were increased to 0·1 to 0·2, which is the normal acidity of the gastric contents in full digestion. E. Hirschfeld[2] also found that 0·07 to 0·08 per cent of hydrochloric acid was sufficient to stop the lactic acid fermentation due to the bacillus acidi lactici (Hueppe), or the fermentation occurring in sour milk.

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[1] *Compt. rend. Soc. de Biol.*, Paris, tome xli. p. 25.

[2] *Ibid.* tome xli. p. 86.

The exact conditions, as far as are known, in which a deficiency of hydrochloric acid is formed during digestion in the stomach are discussed later (Chapter IV.). It is at this point only necessary to state that the two conditions which predispose to the growth of bacteria in the stomach are (1) *the delay of food*, and (2) *a deficiency of hydrochloric acid.*

The following kinds of bacterial fermentation may occur in the stomach contents:—

1. *Acid fermentation.*
2. *Alcoholic fermentation.*
3. *Putrefaction.*

The albuminous substances, the carbohydrates, and the fats, with which there is always an appreciable quantity of fatty acids, are all capable of being decomposed by bacteria in the stomach with the formation of products which are not only of less nutritive value to the organism than the products of the normal fermentations of the alimentary tract, but are in many instances actually harmful.

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[1] Abstract in Maly's *Jahresbericht*, Bd. xx. p. 259.

[2] *Ibid.* Bd. xx. p. 260.

Albuminous substances are decomposed both inside and outside the body by pathogenous organisms, the products being an important element in the pathology of infective disorders. They are also decomposed by putrefactive organisms, rarely in the stomach, more commonly in the intestines. But little is known of the action of bacteria on neutral fats. By far the most important food-stuffs which act as a food for bacteria in the stomach are the carbohydrates; starch, dextrin, glucose, lactose, maltose. Unaltered starch and dextrin, glucose and lactose are taken into the stomach with the food, while some dextrin and maltose are formed during salivary digestion. Maltose is the least readily fermentable of these bodies. Carbohydrates, unlike albuminous substances, do not putrefy, but the result of the action of bacteria on them is to produce acids and alcohols.

Acid Fermentation.—This is the most important bacterial fermentation occurring in the stomach, and during the process acid bodies are formed, sometimes in great abundance, from the carbohydrates of the food. According to the nature of the acid formed, two kinds of fermentation are distinguished, the *lactic acid* and the *butyric acid* fermentation.

Lactic Acid Fermentation.—There are many micro-organisms which form lactic acid from carbohydrates. As many perhaps as fourteen different species have been obtained, some from the air and some from decayed teeth; but there is one which is *par excellence* called the *bacillus acidi lactici*, the action of which was first described by Pasteur, although Lister was the first to obtain it in a pure form. This bacillus is the common cause of the souring of milk, and is found also in decomposed beet juice and in sour bread. According to Hueppe, it is observed as short thick cells, 1 to 1·7 μ long, and 0·3 to 0·4 μ broad. It usually occurs united in pairs, and in milk it forms spores.

For its growth free oxygen is necessary, it is therefore aërobic. It grows readily on gelatine and in various liquid media.

At a temperature of 35° to 42° C. it forms an abundance of lactic acid when grown in solutions of milk-sugar, cane-sugar, dextrin and mannite; carbonic acid being also evolved. When a certain amount of lactic acid is formed, which Hueppe says is about 0·8 per cent, the action stops, to begin again if the lactic acid be neutralised.

The bacillus makes milk gelatinous in fifteen to twenty-four hours, although it does not peptonise the casein.

Starch is converted by it into sugar, from which lactic acid is then formed. The chemical process of the formation of lactic acid from sugar is not properly understood. Bacterial fermentations are not simple processes; they produce in many instances one chief product, with several bye-products. Representing the lactic acid fermentation by a chemical equation, the transformation has been stated as follows :—

$$\underset{\text{Lactose.}}{C_{12}H_{22}O_{11}} + \underset{\text{Water.}}{H_2O} = \underset{\text{Lactic acid.}}{4\ C_3H_6O_3}.$$

Or,

$$\underset{\text{Glucose.}}{C_6H_{12}O_6} = \underset{\text{Lactic acid.}}{2\ C_3H_6O_3}.$$

These equations do not allow for any of the carbonic acid that is formed, but this may be accounted for by a further fermentation which really does occur, viz. the transformation of the lactic acid into butyric acid with the formation of carbonic acid and of hydrogen :—

$$2(C_3H_6O_3) = C_4H_8O_3 + 2\ CO_2 + 2\ H_2.$$

From the point of view at which we are studying these changes, viz. that of disease, it is sufficient to emphasise the fact that the lactic acid fermentation of carbohydrates in the stomach leads to the formation of a large amount of lactic acid and the evolution of carbonic acid gas.

Butyric Acid Fermentation.—There are many bacteria which form butyric acid from carbohydrates, and it is difficult to say which of them is the one that usually produces the butyric acid during the fermentation of food in the stomach. Pasteur first described a *bacillus butyricus*, and Prazmowski, Hueppe, and others have each described bacilli with similar powers and of sufficient activity, as also to be called *bacillus* *butyricus.* Most of these bacilli only grow in the absence of oxygen—they are anærobes; that described by Hueppe was an aërobic micro-organism.

According to Flügge, the bacillus butyricus described by Prazmowski is the one probably identical with that described by Pasteur and by Van Tieghem.

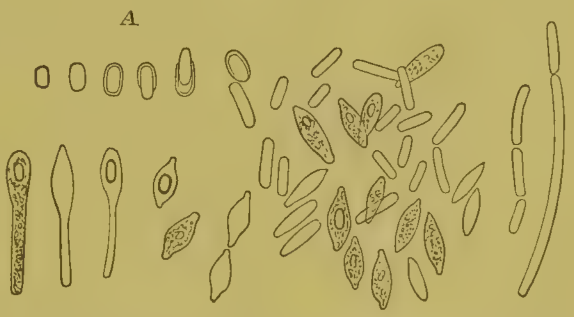

FIG. 14.—Bacillus butyricus, × 1020. Simple rods are seen; also swollen and spindle-shaped spore-bearing bacilli. A, a spore germinating. (Prazmowski.)

Bacillus Butyricus (Fig. 14) (Prazmowski).—This bacillus occurs widely distributed, and is found in decaying vegetable infusions, in old cheese and in milk kept a long time. It is the cause of the butyric acid fermentation of milk. It consists of rods, 3 to 10 μ in length and about 1 μ in breadth; actively mobile, and forming unsegmented threads and spores. It becomes blue on the addition of iodine. Oxygen interferes with the growth of the bacillus, which is an anærobe. When grown in solutions of carbohydrates, such as starch. dextrin, and cane-sugar and dextrin, it forms a large quantity of butyric acid, and produces an evolution of carbonic acid and of hydrogen. Lactose must be first hydrated before the bacillus acts upon it. According to Tappeiner, this bacillus also decomposes cellulose with the formation of marsh gas (methane, CH_4), carbonic acid, and sulphuretted hydrogen, or of only hydrogen and carbonic acid.[1]

A third important chemical action of the butyric acid bacillus is the transformation of lactic acid and lactates into butyric acid with the formation of carbonic acid and hydrogen as shown in the formula on p. 74. Thus, in sour milk which has undergone the lactic acid fermentation this bacillus is capable of transforming the lactic acid into butyric acid, a change which takes place in the stomach contents in disease.

Sarcina (Fig. 15).—The exact *rôle* which sarcina plays in fermentation in the stomach is not clearly known; it is found only in cases of prolonged delay of food in the stomach, with dilatation and advanced fermentation, in which besides acid fermentation the alcoholic fermentation is progressing.

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[1] The bacillus amylobacter (Van Tieghem) was shown by its discoverer to have this action and to be identical with the bacillus butyricus of Pasteur.

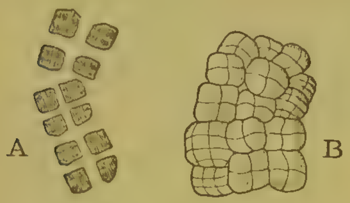

FIG. 15.—Sarcina ventriculi. A, × 600, seen in one plane; B, × 650, diagrammatic, showing the appearance of cotton-bales. (Flügge.)

Goodsir, who first (1842) described the sarcina, found it in the stomach contents in man and animals, and also in the vomit. It possesses a characteristic form, viz. that of corded bales of goods, and is grouped together in very characteristic masses. Goodsir's *sarcina ventriculi* consists of colourless or brownish cells, 2·5 μ (1/100 to 1/1000 inch) in diameter arranged in groups of eight, which are also united into larger masses. Attempts to cultivate the sarcina ventriculi have not been very successful;[1] but the experiments which have been performed show that the sarcina probably produce an acid during their growth.

Besides the sarcina ventriculi there are other well defined forms of sarcina which possess the same general arrangement of cells, but differ in size and in colour. A yellow sarcina, which commonly exists in the air, is named *sarcina lutea*. The cells are smaller than those of the sarcina ventriculi, viz. 1 μ in size, and grow readily on gelatin, agar, and potato. An orange coloured sarcina, *sarcina aurantiaca*, observed in Koch's laboratory, slowly liquefies gelatin. There is, moreover, a *sarcina alba*, and there are possibly other forms which have not been thoroughly studied; but none of these have been found in the stomach contents, and their relation to sarcina ventriculi is unknown.

2. *Alcoholic Fermentation.*—Small quantities of alcohol are formed by the action of bacteria on the carbohydrates, but the amount is quite insignificant in proportion to the quantity of the other products, viz. acid bodies, which are produced. In the alcoholic fermentation of carbohydrates the reverse holds good; alcohol is the chief product, while acids

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[1] See Falkenheim, *Arch. f. exper. Path. u. Pharmakol.*, Leipzig, 1885, Bd. xix. p. 339.

are only bye-products. Alcoholic fermentation takes place from the action of yeast, of which the two most active are beer and wine yeast. The latter (saccharomyces ellipsoideus) is widely distributed in the air (Fig. 16). Glucoses (dextrose, lævulose, lactose) and maltose are the carbohydrates most readily acted upon; and starch, gum and cane-sugar must be converted into glucose before any fermentation occurs.

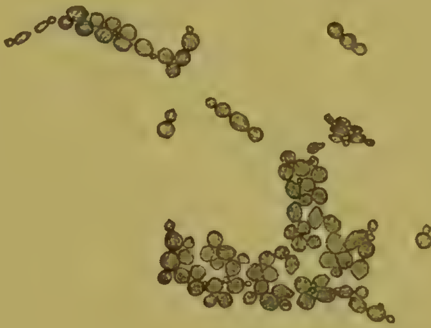

FIG. 16.—Yeast. From a photograph, × about 500. The preparation was made from a culture obtained from the contents of a dilated stomach (p. 143).

The products formed by yeast from these carbohydrates are very complex: alcohol is the chief, but other alcohols (glycerin, methyl-alcohol), succinic and acetic acids, and carbonic acid, are constantly found.

The stomach is not very favourable to the development of the alcoholic fermentation; the temperature is too high. The most favourable temperature for the fermentation is 25° C., while that of the stomach is at least ten degrees higher.

In the stomach contents in advanced bacterial fermentation, not only are sarcina and saccharomyces present, but also various forms of cocci and bacilli.

3. *Putrefaction.*—There is evidence that in rare cases putrefaction occurs in the stomach, not only when actually decomposed articles of food are eaten, but in conditions of prolonged delay of food, and of its fermentation in the organ.

Carbohydrates and fats do not putrefy, and the putrefactive process is one by which numerous kinds of bacteria break up albuminous bodies with the formation of many different kinds of products.

If an infusion of meat, for example, be exposed to the air numerous bacteria will after a time be found to be growing in the liquid, and the proteid bodies present will be found decomposed. The bacteria producing this change are very numerous, and in such an infusion as that named would be of various kinds: they are all classed under the heading of "putrefactive" micro-organisms. Some of these are aërobes, *i.e.* require oxygen for their growth; others are anærobes, *i.e.* grow most rapidly in the absence of oxygen. In such an infusion as we have supposed the aërobic micro-organisms develop first, forming a scum on the surface; after the oxygen in the liquid has been exhausted the anærobic develop. Some of these bacteria have been isolated and their products studied in pure cultivations. Our knowledge of them is not at present sufficient to ascribe to each individual bacterium its exact *rôle* in the chemistry of putrefaction. It is much more important from the standpoint at which we are viewing putrefaction, to discuss the kinds of chemical substances which may be formed during the putrefactive process.

(1) There are certain gases, such as carbonic acid, hydrogen, and nitrogen, sulphuretted hydrogen and marsh gas; (2) there are acids of the fatty acid series, such as formic, acetic, butyric and valerianic; (3) other acid bodies, such as lactic, succinic, glutamic and aspartic acids; (4) ammonia and amine bases, such as trimethylamine; (5) foul-smelling, and inodorous bodies belonging to the aromatic series, indole, skatole, tyrosine, etc.; (6) the so-called "ptomaines"; and (7) albumoses. The albumoses are the first products in the change which the proteids undergo; all the others are later products. From the pathological point of view these chemical bodies may be divided into three classes:—

1. The acid bodies which increase the acidity of the stomach contents and thus diminish the activity of the gastric juice.

2. The gases which are eructated and which are sometimes foul-smelling, as when they contain sulphuretted hydrogen, and inflammable as when marsh gas (CH_4) and hydrogen predominate.

3. The poisonous products. Some of these which have been studied belong to the class of ammonia compounds (amines). Their *rôle* in producing symptoms in disease of the stomach is as yet not obvious. Still, when a patient has a putrefying fermenting mass in the stomach, as occasionally happens, some of the symptoms observed are to be ascribed to the absorption of poisonous bodies from the stomach contents.

The result as regards the stomach of the various changes of the food brought about by micro-organisms may be summarised as follows:—

1. The acidity due to organic acids is increased by the formation of lactic, butyric, and acetic acids chiefly. This acidity is inimical to the efficient action of the pepsin which is most active in the presence of hydrochloric acid, and to the secretion of the acid of the gastric juice (Chapter I.). Organic acidity of the stomach contents is chiefly due to the lactic acid and butyric acid fermentation, to some extent to the growth of sarcina and to the occurrence of the alcoholic fermentation.

2. Gases are produced, chiefly carbonic acid and hydrogen (acid fermentations and putrefaction), or sulphuretted hydrogen and marsh gas in rarer cases (due to putrefactive processes).

3. Perhaps the production in some cases of poisonous bodies in the stomach contents may occur from putrefaction.

In the majority of instances the acid fermentations are the chief processes in the stomach, and these destroy the carbohydrates, so that they are not assimilated by the organism, and are wasted as food.

CHAPTER IV.

PATHOLOGY OF INDIGESTION OF FOOD—(*Continued*).

FOOD as a cause of its own non-digestion by the stomach and the fermentative changes which may occur in the stomach contents have been discussed in the previous chapter, and an indication has been given of some of the changes in the functions of the stomach which occur in disease. The normal processes of digestion are dependent on the secretion of gastric juice, on the movements of the organ, and on the absorption. In disease these functions are variously affected, either by an increase or a diminution of activity, and although one particular function may be affected more than another in individual cases, yet all the functions are altered from the physiological standard if only one appears to be affected. It will be most convenient to consider the subject under the following headings:—

1. The acids of the stomach in disease.
2. The gases of the diseased stomach.
3. The digestive activity of the gastric juice.
4. The movements of the diseased stomach.
5. Absorption by the stomach in disease.

1. *The Acids of the Stomach.*—Physiological and chemical investigation has settled the question as to the acid which is the cause of the acidity of the gastric juice. The researches of Prout, of Bidder and Schmidt and of others, have conclusively shown that free hydrochloric is not only present in the gastric juice, but it is the acid which must be present in order that gastric digestion should be efficient. It is true that

lactic acid may also be present in normal digestion, especially in the early stages, and this occurs particularly after the ingestion of carbohydrate and other food containing lactates. Its place, however, is soon taken by hydrochloric acid, and this in normal digestion remains the chief acid (Chapter I.).

In disease, by an examination of vomited matters and of the stomach contents removed during digestion (Chapter V.), three conditions of acidity have been found.

1. Hyperacidity of the stomach contents, due to hydrochloric acid.
2. Diminished acidity, due to a deficiency in the amount of hydrochloric acid secreted.
3. Increased acidity, due to organic acids (lactic, butyric, acetic).

1. *Increase in the amount of hydrochloric acid secreted by the stomach; hyperacidity due to hydrochloric acid.*—In such cases there is a secretion of very acid gastric juice during digestion, and this may continue after digestion is completed. There may thus always be some acid liquid and remnants of the food in the organ. Individual cases, however, differ: in some, a large amount of acid is secreted in half an hour to one hour after a meal, and this continues during the process of digestion, ceasing, however, towards the end; in other and more severe cases, the secretion continues, even after the mass of the food has been expelled into the duodenum. Hyperacidity due to hydrochloric acid occurs chiefly in functional disorders of the stomach, although it may be the first stage of catarrh, of which it is one of the causes: it occurs also in some cases of chronic ulcer, but bears no known pathological relation to the formation of an ulcer. The prime factor in the causation of hyperacidity is irritation of the gastric mucous membrane. The results of irritation of a living tissue depend not only on the strength of the irritant, but on the condition of the nervous system. In cases, for example, of irritability of the nervous mechanism, a slight stimulus may produce a good effect. In stomach disorder, hyperacidity may be directly due to the breach of dietetic rules, food and food accessories (especially alcohol) acting as irritants (p. 68), or it

may occur when the nervous mechanism is disordered, as in cases of "nervous dyspepsia" (neuroses of the stomach). In ulcer both factors probably play a part in its production. The direct observations which have been made on the occurrence of hyperacidity in disease will now be considered.

Testing the acidity of the stomach contents in two cases by means of a solution of methyl-violet, Reichmann[1] found that there was a large amount of hydrochloric acid present even when the patients were fasting; the acidity reckoned as HCl was as much as from 0·306 to 0·32 per cent, the normal being about 0·2 per cent. Riegel obtained similar results in certain cases.[2] Jaworski examined the stomach contents of 222 persons during fasting and found that in 179 (81 per cent) the contents were acid, and that this acidity was due to hydrochloric acid.[3] In all the cases mentioned there was no evidence of organic disease of the stomach—ulcer or carcinoma.

In chronic ulcer of the stomach, there is in some cases hypersecretion of hydrochloric acid, as much as 0·226 to 0·325 per cent being found in the stomach contents (Rothschild).[4] Korczynski and Jaworski examined twenty-four cases of ulcer with a view of testing the secretion of acid, and found that there was hyperacidity of the stomach contents during digestion, and that even in the fasting stomach the contents were acid. They concluded that a bleeding round ulcer could not extend in the absence of hydrochloric acid, a conclusion which is certainly not warranted by the facts known about ulcer of the stomach.[5] It is, however, not in all cases of ulcer of the stomach that there is hyperacidity due to hydrochloric acid; in individual cases the acidity may be diminished, and may be due to both hydrochloric acid and to organic acids. This has been shown by Cahn and von Mering,[6] who investigated the acidity of the stomach contents in four cases of ulcer of the stomach. The results obtained are shown in the following table, the diet being given in the first column :—

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[1] *Berl. klin. Wchnschr.*, No. 2, 1884.

[2] *Ztschr. f. klin. Med.*, Berlin, No. 11, 1886.

[3] *Wien. med. Wchnschr.*, Bd. xlix., 1886. See also Martius and Lüttke, "Die Magen-Säure des Menschen."

[4] Inaug. Dissert. *Strasburg*, 1886.

[5] *Deutsche med. Wchnschr.*, Leipzig, Bd. xlvii.

[6] *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxxix. p. 248, 1886.

	Percentage of HCl in stomach contents.	Percentage of lactic acid.	Percentage of volatile acids.
Case 1. Male, æt. 46. Round ulcer; hæmatemesis and pain, atony of stomach. Diet: milk and gruel. Stomach contents removed five hours after food . .	0·203	0·095	0·008
Case 2. Male, æt. 28. Ulcer of stomach and dilatation. Mixed diet	0·131– 0·197	0·027– 0·099	0·024– 0·046
Case 3. Male, æt. 46. Long-standing ulcer of stomach, with slight dilatation; pain, previous hæmatemesis, great anæmia. Stomach contents removed on entering hospital. Mixed diet. . . .	0·066	0·014	0·007
Case 4. Male, æt. 45. Long-standing ulcer of the stomach, with hæmatemesis and pain. Dilatation. Diet: coffee, milk and eggs	0·162	0·149	0·006

These results show that the acidity of the stomach contents may be normal or below the normal, especially in long-standing cases, and in those in which there is dilatation of the organ or great anæmia (p. 84 *et seq.*). The acidity, therefore, varies greatly in chronic ulcer; and it may be concluded that in some cases the acidity of the stomach contents during digestion is normal; in others it is above the normal; and in some, especially those of long-standing, there is great defect of digestion due to a diminished secretion of gastric juice.

2. *Diminution in the Amount of Hydrochloric Acid secreted by the Stomach.*—Diminution in the amount of hydrochloric acid secreted by the stomach plays a greater *rôle* in disordered digestion than excessive secretion. There are three chief causes of the diminution in the secretion of hydrochloric acid, viz. :—

1. Those due to long continued irregularities in the diet, whether in the amount of food taken, in the quality of the food, or the amount of food accessories, or to any of the other conditions which have already been fully discussed. In these cases there is frequently a first stage of hyperacidity: irritation leading first to increased, and then to diminished function.

2. Those due to actual anatomical changes in the stomach walls, either directly affecting its secreting apparatus (congestion, catarrh, atrophy) or directly and indirectly affecting the glandular structures, as in cancer of the stomach.

3. Those due to conditions outside the stomach which affect the functions, either through the nervous system or by the quality of the blood supplied to the organ. Such conditions are irregularities in the mode of life; the state of fever; chlorosis and other forms of anæmia (*e.g.* pernicious anæmia); chronic wasting diseases (cancer and other new growths); acute infective diseases (tuberculosis, typhoid fever, measles, malaria, scarlet fever), during the acute stage, in the chronic stage and in or after the period of convalescence.

Deficiency in the secretion of hydrochloric acid may be present therefore in both functional and organic diseases of the stomach. It is difficult, however, in each of the individual cases that have been investigated to ascribe the exact part played by food, by anatomical changes in the gastric mucous membrane, and by general diseases in the production of the disordered function. In the majority of the cases the conditions are not simple. The food itself leads to gastric catarrh, and primary gastric catarrh is aggravated by the amount and character of the food taken: with gastric catarrh and with stomach disorders due to general disease, there may be anatomical changes in the glandular structure of the organ, there may be dilatation of the organ, and there may be fermentation of the stomach contents: and functional disorders may be due to the effect of the food and of the general condition of the body. Bearing these facts in mind, the results of the investigation of the amount of hydrochloric acid present in various functional and organic diseases of the stomach may be mentioned.

In three cases of uncomplicated "*chronic dyspepsia*" Leube[1] found by his method of investigation (Chapter V.) a well-marked deficiency of hydrochloric acid. In chronic dyspepsia with dilatation of the stomach (two cases) he also found a deficiency in the secretion of hydrochloric acid. In seventeen cases of dilatation of the stomach without ulcer or carcinoma, Kredel found hydrochloric acid present in all; although the methods he used were not so accurate as Leube's or as Cahn and von Mering's. These last observers estimated the acidity in three cases of dilatation of the stomach with the following results:—

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[1] *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxxiii. p. 19.

	Percentage of HCl in stomach contents.	Percentage of lactic acid.	Percentage of volatile acids.
Case 1. Male, æt. 43, with chronic catarrh and great dilatation of the stomach. Diet: meat, spinach, bread. First examination on entering hospital . . .	0·299	0·063	0·006
Case 2. Male, æt. 62, with dilatation of stomach due to atony. Stomach repeatedly washed out. Mixed diet. Stomach contents examined four hours after last meal	0·255	0·054	—
Case 10. Male, æt. 10, with great dilatation of stomach following on left-sided pleurisy	0·166	0·065	0·080

These results are very suggestive as showing that even in dilatation of the stomach a large amount of hydrochloric acid may be secreted, and that also in atony of the stomach in old age the acid may be present in normal quantity in the contents of the organ.

Leube examined the stomach contents in seven cases of "nervous dyspepsia"; in three, the acidity was normal, in four it was deficient, markedly so in two of them. Cahn and von Mering found in a girl aged 22 years, suffering from hysterical vomiting, and fed on beef-tea, meat, bread, and rice-milk, that three-quarters of an hour after food the contents of the stomach contained 0·116 per cent of hydrochloric acid and 0·057 per cent of lactic acid. Nervous dyspepsia and neuroses of the stomach are, however, not infrequently characterised by the secretion of a varying amount of hydrochloric acid; at one time in excess, at another in deficient quantity.

In *acute catarrh* the secretion of gastric juice greatly diminishes, becoming almost nil, the stomach being quite incapable of digesting and manipulating a test meal of soup, meat, and bread. Pepsin may be quite absent from the stomach, or it is present in such small quantity that, with the diminished quantity of hydrochloric acid, it is useless for the digestion of solid food. During convalescence, the pepsin secretion recovers itself sooner than the acid, as is shown in the analysis of the vomit given in Chapter V. p. 141. In chronic catarrh of the stomach, as far as the question has been investigated, hydrochloric acid is present in the stomach, but is greatly diminished in quantity; and some cases are preceded by a stage of hyperacidity (p. 81). In what Jaworski calls "atrophic stomach catarrh," the acid is absent. This is probable, and indeed the conclusion can be extended to all cases of atrophy of the mucous membrane of the stomach (Fenwick), and is most marked in those cases of atrophy which result from the action of the mineral acids and caustic alkalies on the gastric mucous membrane. In these last cases, the corrosive poison not only by its action destroys some of the mucous membrane, but sets up a chronic congestion and fibrosis which leads to further degeneration (Chapter IX.). In the early stage of the disease in such cases, hydrochloric acid may be present in normal quantity. This occurred in the case of a girl, aged 24 years, who took crude hydrochloric acid; subsequently there developed great dilatation of the stomach with stenosis of the pylorus. An examination of the stomach contents when the patient was on a milk diet showed a percentage of 0·15 of hydrochloric acid and 0·2 of lactic acid (Cahn and von Mering). In this case there were a large number of bacteria in the stomach.[1] In *albuminoid degeneration* of the stomach, hydrochloric acid has been found completely absent (Edinger),[2] but it is not always so. In a case of pulmonary tuberculosis in a patient aged 28 years, with albuminoid degeneration of the abdominal organs and of the gastro-intestinal tract (verified post-mortem), the stomach contents were examined twenty-six hours before death, and 0·24 per cent of hydrochloric acid, with 0·054 per cent of lactic acid and 0·042 per cent of butyric acid, were found (Cahn and von Mering).

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[1] W. de Bary, *Arch. f. exper. Path. u. Pharmakol.*, Leipzig, Bd. xx. p. 249.

[2] Edinger used only the colour reactions (chiefly methyl-violet) in testing for free hydrochloric acid. His results are therefore not altogether reliable. See Chapter V.

The conditions of acidity of the stomach in *chronic ulcer* have already been discussed (p. 82).

In *carcinoma of the stomach* varying results have been observed. Van der Velden[1] tested the stomach contents in eight cases of cancer of the pylorus by means of the methyl-violet reaction, and found that no free hydrochloric acid was present. Kredel[2] examined nineteen cases of cancer of the stomach with dilatation and found free hydrochloric acid absent; he used the methyl-violet and the tropœolin tests. Riegel[3] obtained the stomach contents in cases of carcinoma one hour after food, and found that for long periods (months) there may be no free hydrochloric acid in the stomach of such patients. Korczynski and Jaworski[4] state that in most cases of cancer of the stomach hydrochloric acid is absent; and in one case investigated by Leube the acid was also deficient. On the other hand, Cahn and von Mering by a more accurate method of investigation found that free hydrochloric acid was present in most cases of cancer of the stomach, and that its absence was the exception. This is most in accord with a rational view of the disease. In cancer elsewhere than in the stomach, *e.g.* in the œsophagus, hydrochloric acid is present in the gastric juice in normal amount, as I have myself observed in a patient on whom gastrostomy had been performed. There seems no adequate reason why a localised growth at the pyloric end of the stomach, a region in which the hydrochloric acid is not secreted, should affect the secretion of the acid by the glands of the cardia: but in the later stages of the disease when there is atrophy of the glands, either primary or secondary, or general fibrosis of the organ, there is great diminution in the secretion of gastric juice, and its digestive activity is practically nil.

Cahn and von Mering investigated the acids in six cases of carcinoma of the pylorus during life, in one case after death, and in one case in which the diagnosis was doubtful.

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[1] *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxiii. p. 369.

[2] *Ztschr. f. klin. Med.*, Berlin, Bd. vii. p. 592.

[3] *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxxvi. p. 100.

[4] *Deutsche med. Wchnschr.*, Leipzig, No. 47, 1886.

PERCENTAGE OF ACIDS IN THE STOMACH CONTENTS IN CASES OF CARCINOMA VENTRICULI.

	Percentage of HCl in stomach contents.	Percentage of lactic acid.	Percentage of volatile acids.
Case 1. Male, æt. 48. Hard tumour along contour of stomach, cancerous peritonitis, great cachexia. *Post-mortem:* colloid cancer of pylorus, of antrum pylori and part of the great curvature; cancerous nodules in peritoneum . .	0·029 0·096 0·051	0·162 0·350 0·547	0·015 0·036 0·043
Case 2. Male, æt. 63; with very large tumour of pylorus and great dilatation of the stomach	0·140	0·630	0·012
Case 3. Female, æt. 52, with hard tumour of pylorus and great dilatation of the stomach. *Post-mortem:* alveolar cancer of the pylorus and of a large part of the lesser curve . . .	0·097 ...	0·090 0·387	0·045 0·014
Case 4. Male, æt. 52, with great anæmia and pain, great dilatation of the stomach and a pyloric tumour the size of the fist	0·001 0·118	0·234 0·109	0·002 0·003
Case 5. Male, æt. 36, with an immovable pyloric tumour, and a dilated stomach reaching to the symphysis and exhibiting peristalsis	0·044 0·099	0·117 0·108	0·090 0·120
Case 6. Female, æt. 22, greatly emaciated and with enormous dilatation of the stomach caused by a cancer of the pylorus (Gastro-enterostomy successfully performed by Prof. Lücke) .	0·054	0·261	0·054
Case 7. Male, æt. 34, with stenosis of pylorus and great dilatation of stomach. No tumour felt. Diagnosis not absolutely certain	0·041	0·540	0·025

Many clinicians accepting van der Velden's original dictum have asserted that the absence of hydrochloric acid in the stomach contents is a trustworthy diagnostic sign of carcinoma ventriculi in doubtful cases. The observations just quoted demonstrate that this is not so, and that as Cahn and von Mering say, "In carcinoma pylori the presence of hydrochloric acid is the rule, its absence the exception." A study of the table just given will show that in some cases only traces of the acid were found (Case 4, first observation), but that in most cases the percentage of acid was appreciable (Cases 2, 3, 4, and 5), while different observations on the same patient showed that a trace of acid might be found on one occasion and nearly a normal quantity on another (Case 4). The general conclusion therefore is that in carcinoma of the pylorus, the amount of hydrochloric acid secreted is below the normal, and that it varies greatly at different times even in the presence of food in the stomach. The other acids found in the cases observed will be the subject of discussion when the organic acids of the stomach are considered (p. 93); they are chiefly the products of fermentation of the food, and are associated with the condition of delay of food in the stomach and dilatation of the organ.

The secretion of acid by the stomach is affected in general diseases or general pathological states, as well as in cases where the stomach itself is the primary seat of disorder or disease. The relation of functional disorders of the stomach to other diseases of the body will be fully discussed when the clinical history of such cases is considered. It will be sufficient at this point to note that the occurrence of functional disorder of the stomach in a particular disease may be accidental and not necessarily a question of effect and cause; or it may be a direct effect of the disease.

In most *anæmic conditions* it is probable that the amount of hydrochloric acid secreted is deficient, although extensive and accurate observations have not yet been made. In cases of anæmia with chronic dyspepsia Leube found a great deficiency of hydrochloric acid after a test meal; and in one case of pernicious anæmia, fed on milk and beef-tea, Cahn and von Mering found that three-quarters of an hour after a meal the stomach contained no hydrochloric acid, but a large amount of lactic acid (0·219 per cent) and a small amount of volatile acid (0·05 per cent). The observation was made three months before death. In chlorosis, the most common form of anæmia, accurate observations have not been made.

In *infective fevers* the amount of hydrochloric acid present in the stomach no doubt varies to a great extent from time to time. Some accurate observations have been made regarding the presence of hydrochloric acid in the stomach in such diseases. Manassein[1] made animals febrile by injecting putrid material into them, and found that the gastric juice had no digestive properties unless hydrochloric acid was added to it.

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[1] *Virchow's Archiv*, Bd. lvi. p. 413.

In three cases of *typhoid fever* examined by Cahn and von Mering the hydrochloric acid was greatly deficient, and in two, as seen in the table, there was an excess of lactic acid.

	Percentage of hydrochloric acid.	Percentage of lactic acid.	Percentage of volatile acids.
Case 1. Typhoid fever, with remittent fever. Diet: beef-tea with egg, milk . .	0·058	0·144	0·012
Case 2. Relapsed typhoid, with high fever. Diet: cocoa, milk, beef-tea . .	0·006	0·203	0·030
Case 3. Typhoid fever in a girl with cerebral tumour; continued fever. Diet: milk and cocoa	0·0	0·09	0·045

The presence of lactic acid in these cases is no doubt to be ascribed to the sarcolactic acid present in the beef-tea, and not to the occurrence of bacterial fermentation.

In two cases of *malaria*, Leube observed severe dyspeptic symptoms independent of the ingestion of food, which were not due to a prolongation of the digestion or to an insufficiency in the activity of the juice, as tested by his method. These cases recovered rapidly with quinine.

In *tuberculosis*, more particularly tuberculosis of the lungs (phthisis pulmonum), numerous observations have been made. In tuberculosis of the lungs, indigestion of food is frequently observed, both before the signs of tuberculosis are manifest, and during the progress of the disease. It has been stated that in pulmonary tuberculosis, hydrochloric acid is greatly deficient or absent from the stomach, but this statement, like the similar one with regard to carcinoma ventriculi, is too decided, and does not apply to the majority of cases. Klemperer[1] found in the cases examined by him no diminution in the secretory activity of the stomach, and a similar result was obtained by Fr. Schetty in the twenty-five cases of the disease which he examined.[2]

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[1] *Berl. klin. Wchnschr.*, 1889, No. 11.

[2] *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xliv. pp. 219-243.

Schetty tested the digestive activity by giving the patient the whites of two hard-boiled eggs with four to six ounces of water, and emptying the stomach in an hour. In all cases, the stomach contents were acid, and the acidity was due to hydrochloric acid (the phloroglucin-vanillin test was used, see Chapter V.). Not only was the secretion of hydrochloric acid in the majority of the patients not diminished, but in some it was actually increased. A diminution in the amount of acid was found in advancing cases and in those with morning fever.

Schetty's results do not express the condition which is found in all cases or even the majority of cases of pulmonary tuberculosis; the degree of acidity cannot be tested with any accuracy by means of the colour tests he employed. O. Brieger[1] estimated the acidity in sixty-four cases of pulmonary tuberculosis, making 300 experiments in all. His method was to give a "test-breakfast" (Chapter V.), and to estimate the acidity of the stomach contents after removal. The acidity due to hydrochloric acid varied between 0·05 per cent and 0·19 to 0·24 per cent. In severe cases of the disease, he found that the chemical processes of digestion were normal in only 16 per cent, deficient in the greater number, and in 9·6 per cent completely deficient; and that the changes in digestive power had no relation to the febrile state of the patient. These results have not been confirmed in all particulars by other observers. C. H. Hildebrand[2] examined the acidity in forty cases of the disease and found that in some cases hydrochloric acid was present and in some absent. In the cases in which it was absent there was fever, and no hydrochloric acid was found during the febrile period; the cases in which the acid was present were afebrile. It is probable that these results are nearer the truth than those previously quoted. The occurrence of indigestion of food in pulmonary tuberculosis is probably not a simple question. In some cases, but by no means in the majority, the symptoms of dyspepsia precede the evident signs and symptoms of the disease, and in such cases the stomach condition may be simply looked upon as one of the predisposing causes of the disease. In cases where pulmonary tuberculosis has actually developed, the patient is suffering from a slowly progressing disease in which there is fever, wasting, and an anæmic condition. It is therefore only a natural sequence that the secretory and other functions of the stomach should be affected by the condition of fever and anæmia produced by the disease, and that this effect would be more marked as the disease advanced. It is, however, a remarkable fact that in many cases of advancing pulmonary tuberculosis the digestion is normal; food in excess is in some cases eaten without discomfort, and large quantities of fat are eaten with benefit. Deficient activity of the stomach, a condition of functional gastric insufficiency, is not the only disorder of the organ associated with pulmonary tuberculosis: there may be hyperacidity during digestion, due to gastric irritation; or there may be chronic catarrh of the organ, producing degeneration of the glandular structures and organic gastric insufficiency; or albuminoid degeneration of the mucous membrane (p. 86). These four conditions must be borne in mind in discussing the alteration of stomach function in pulmonary tuberculosis. There is in fact no evidence to show that there is such a pathological condition as "phthisical dyspepsia," *i.e.* an indigestion of food produced by the development of the bacillus tuberculosis. Clinically, dyspepsia in pulmonary tuberculosis is an important condition; pathologically, it has no very direct relation to the disease.

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[1] *Deutsche med. Wchnschr.*, Leipzig, 1889, No. 14.

[2] *Ibid.* 1889, No. 15.

Summary.—As regards our knowledge of the amount of hydrochloric acid present in diseased conditions of the stomach, and in diseased conditions generally, it may be said that in some cases (as in cases of irritation, of nervous dyspepsia, and of ulcer of the stomach) hydrochloric acid is secreted by the stomach in excess, and that in other cases there is a diminution in the amount of acid secreted. In prolonged cases of ulcer, in chronic catarrh, and in many cases of carcinoma ventriculi, the secretion of acid is diminished; and in acute catarrh it is absent. In certain febrile conditions (as typhoid fever), in pernicious anæmia, and in tuberculosis of the lungs, the acid may be greatly diminished or even absent during digestion. The presence or absence of hydrochloric acid in the stomach contents is of great importance in disease. Its presence in excess during digestion may ensure rapid digestion of the food, but it induces symptoms which are often severe, and it may lead to inflammation of the organ. Without its presence in a certain proportion digestion cannot progress normally, even if lactic, butyric, acetic, or any other organic acid is present in the stomach contents, as none of these can take its place. It is therefore of prime importance to remember that in the majority of cases of functional disorders of the stomach which constitute the greater number of cases of disease of the organ the acid is present, and its presence shows that the glands are still in a state (though a diminished state) of secretory activity, and by treatment can be made to resume their normal condition.

3. *Increased Acidity of the Gastric Contents due to Organic Acids (Lactic, Acetic, Butyric).*—A certain quantity of organic acids is present in the stomach contents during digestion in most healthy people taking a mixed diet of proteids, fats, and carbohydrates in the proper proportion; indeed, certain organic acids or their salts are essential for the needs of the economy, such as acetic, citric, tartaric, and malic acids and their salts, since they not only tend to preserve the alkalinity of the tissues by being converted into carbonates in the body, but are to some extent antiscorbutic. When the salts of these acids (or indeed of all the organic acids under consideration) are taken with the food they are decomposed by the hydrochloric acid of the gastric juice, the acids being in great part set free. Some of these organic acids may be formed in the stomach by decomposition of the food. There are therefore two distinct modes of origin of the organic acids found in the stomach contents: (1) they are taken in with the food, and (2) they are formed from the food in the stomach by bacterial fermentation.

1. In normal conditions a certain amount of acetic acid, citrates and tartrates, not only do no harm, but are actually beneficial; in excess, however, they increase the organic acidity of the stomach and may lead to digestive disturbance. They also in excess render the urine alkaline and increase the alkalinity of the saliva (see p. 36).

An increase of the organic acidity of the stomach contents leads to a delay of the chemical processes of digestion, but it has another effect which is of great importance in disease. When the stomach has for some time been the seat of disorder, or when it is the subject of an acute affection, the increase of the organic acidity, whether due to the vegetable acids (acetic, tartaric, citric) or to the acids formed by fermentation (lactic, butyric, and acetic), acts as an irritant to the gastric mucous membrane, increasing the seriousness of the condition. The occurrence of lactic, acetic, and butyric acids in the stomach during normal digestion and their origin in this condition has already been fully discussed (pp. 31, 62, and 68). It was shown that the lactic acid found in the stomach during the digestion of bread and meat came from the food itself, and was not formed in the stomach.

2. The question now to be considered is the increased acidity of the stomach contents, due to the formation of organic acids as the result of bacterial fermentation. When the acidity of the stomach contents rises to a certain percentage, vomiting occurs, so that part of the knowledge of the hyperacidity due to organic acids has been obtained from the examination of vomited matters, and valuable information has also been obtained of these conditions by removing the stomach contents some time after food.

The hyperacidity due to organic acids may be divided into three classes. In one, the commonest form of hyperacidity, there is a large excess of lactic acid in the stomach contents. In the second class, the excess of acid is due to the presence of butyric acid, and in the third to acetic acid. Excess of lactic acid and of butyric acid is the most important. All these acids may be present in the stomach contents of any particular case where there is bacterial fermentation of the carbohydrates of the food, but as a rule one acid preponderates. All these acids may be present in the food taken; but as a rule they are present only in small amount, and their presence in large amount in the stomach contents is a pathological condition.

An excess of lactic acid in the stomach contents is the result of the lactic acid fermentation of the carbohydrates of the food, due to the growth of the bacillus acidi lactici, or possibly of other bacteria which have been found in the stomach, viz. bacillus lactis aerogenes (see p. 71). An excess of butyric acid is the result of the butyric acid fermentation of the carbohydrates of the food, or of a similar fermentation of the lactic acid formed in the lactic acid fermentation (see p. 74). An excess of acetic acid is the result of the alcoholic fermentation of the carbohydrates in the stomach, and possibly of the action of sarcina, although this is doubtful, or in rare cases perhaps of the action of mycoderma aceti.

It is quite clear that an excess of these acids in the stomach can only be produced by bacterial fermentation; they are not products of digestion by the gastric juice, and the amount taken with the food never approximates the degree of organic acidity found in the cases of disease under consideration. Another point must be clearly borne in mind, viz., that although bacterial fermentation leading to an excess of organic acidity of the stomach most readily occurs in certain conditions of that organ, more particularly in dilatation, whether due to obstruction or not, yet it may occur, although rarely, in cases of functional disorder, and in these cases it is associated with delay of food in the stomach and cessation of the secretion of gastric juice during digestion. It may not occur in individual cases where the conditions are favourable, as in some cases of dilated stomach not due to obstruction. This increase in the organic acidity of the stomach contents is indeed a change in the food taken, which only occurs when there are certain predisposing causes, and it cannot occur unless the bacteria producing it are swallowed with the food or are already in the organ.

The total amount of acidity which may be present in the stomach contents in such cases of bacterial fermentation may be very great. In a case of dilated stomach with the vomiting of a frothy liquid containing sarcina, the total acidity of the stomach contents some hours after food was found equal to 0·48 per cent hydrochloric acid, the acidity being due to free hydrochloric acid, to lactic acid, but chiefly to butyric acid. In a case of indigestion of food of fifteen years' duration, associated with bacterial fermentation and the presence of large numbers of sarcina in the stomach contents, Hassall[1] found the vomited matter to have the following chemical composition :—

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[1] *Lancet*, 1853, vol. i. pp. 338, 362.

PERCENTAGE COMPOSITION OF VOMIT IN A CASE OF BACTERIAL FERMENTATION.

Water	97·582
Free hydrochloric acid	0·080
Free butyric acid	0·138
Yellow fatty matter	0·800
Extract soluble in alcohol	0·242
Gummy extract soluble in water	0·400
Albumen	0·200
Alkaline chlorides and basic sulphates	0·458
Calcium phosphate	0·100
	100·000

In this case butyric acid was the chief acid found, and there was no lactic acid present.

In the case in which Goodsir[1] first discovered the sarcina ventriculi, a careful analysis of the vomited matters was made by Wilson. Hydrochloric acid was found, as well as lactic acid, but acetic acid was present in enormous quantity and formed the greater proportion of the free acids present in the stomach contents. The presence of the acetic acid in such quantity would indicate the occurrence of the acetic acid fermentation or of the alcoholic fermentation.[2]

As regards the presence of lactic acid, the results of the investigation by Cahn and von Mering have been already given in discussing the amount of hydrochloric acid present in the stomach contents in disease. On referring to the table (p. 85) it will be seen that the percentages of lactic acid and of volatile acids found in the three cases of dilatation of the stomach, without obstructive organic disease, were not greater than those which might be found under normal conditions. In the cases of ulcer of the stomach (Table, p. 83) similar observations may be made, except with regard to Case 4, in which the amount of lactic acid was 0·149 per cent. These results must be considered only as meaning that in some cases of simple dilatation of the stomach, and in some cases of ulcer with dilatation, there is not a great increase, or any increase at all, of the organic acidity of the stomach. On the other hand, it is well known clinically that in all such cases as those under consideration bacterial fermentation may occur, and that it may produce a great increase in the organic acidity of the stomach contents.

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[1] "Anatomical Memoirs," 1868, vol. ii. p. 351. The original paper was published in 1842.

[2] For more examples of the analysis of the stomach contents, see Chapter V. p. 141 *et seq.*

In cases of organic obstruction of the pylorus, due to carcinoma ventriculi, very high degrees of acidity of the stomach contents, due chiefly to lactic acid, may be found. Cahn and von Mering's results are shown in the table (p. 88). The highest degree of acidity found by them was in Case 2, in which 0·63 per cent of lactic acid was present; in two other cases (Cases 1 and 7) there was a percentage of 0·54 to 0·547 lactic acid; in the other cases a smaller percentage, but still a high one, was found. In these cases it was pointed out that the amount of hydrochloric acid varied at different times in the same individual; a similar remark applies to the amount of lactic acid present in the same stomach at different times. Thus, in Case 1, three different estimations of the acidity of the stomach contents gave 0·162, 0·35, and 0·547 per cent lactic acid. In Cases 3 and 4 there was a similar variation in the amount of acid present.

2. *Gases of the Stomach in Disease.*—In the healthy stomach, the gases present are nitrogen and oxygen, which are contained in the swallowed air. In some cases there may be an excess of carbonic acid, due to the decomposition of the carbonates of the food by the hydrochloric acid of the gastric juice, or to the decomposition of the carbonates of the alkaline saliva.

In disease the gas eructated is of variable composition, and according to the gases present, two classes of cases may be distinguished: in one the gas being composed of nitrogen, oxygen, and carbonic acid; in the other, of the same gases with hydrogen, and sometimes marsh gas and sulphuretted hydrogen. The first class of cases is due either to the carbonates of the food or of the swallowed saliva, which in some cases of disordered digestion (hyperacidity) is greatly increased (p. 116), and when swallowed leads to the evolution of carbonic acid gas which is eructated; or it is due to the entrance into the stomach of the alkaline fluids of the duodenum which are decomposed by the gastric juice, evolving carbonic acid; or to the entrance of gas from the intestines; or to swallowed air. In hysterical flatulence, the large amount of gas sometimes eructated has been ascribed either to swallowed air or to a diffusion of carbonic acid from the blood. There is some evidence that the second event can occur; but swallowed air is sometimes a cause of the flatulence, and also the entrance of gas into the stomach from the intestine. The commonest cause of eructation of gas is, however, the bacterial fermentation of food which occurs in the stomach and intestine. The formation of carbonic acid and hydrogen by the lactic acid, butyric acid, and alcoholic fermentations has already been described (p. 74 *et seq.*); and in severe cases of flatulence (excluding "hysterical" flatulence) the evolution of gas is due to the occurrence of one or other of these kinds of fermentation. In these cases the gas eructated is composed of nitrogen and oxygen (derived from the air) and a large quantity of carbonic acid, together with varying proportions of hydrogen.

In other, but rare, cases the eructations contain marsh gas (CH_4) and are inflammable. In one such case described by M'Naught, the gas had the following composition: 9·2 per cent of atmospheric air, 56 per cent of carbonic acid (CO_2), 28 per cent of hydrogen (H), and 6·8 per cent of marsh gas (CH_4).

Marsh gas is formed from decaying vegetable matter, and is a product of the decomposition of albuminous substances (p. 78). According to van Tieghem and Tappeiner (p. 75) the butyric acid bacillus is capable of forming from cellulose both marsh gas and sulphuretted hydrogen. This is, however, a result which has not been shown to occur in the stomach, and it is improbable that it does occur. Putrefactive decomposition of proteids may, in rare instances, occur in the stomach as the result of long standing retention of food in cases of dilatation due to organic disease; but it more commonly results from taking as food, articles of diet in a state of incipient decomposition. In some of these cases the eructations contain sulphuretted hydrogen, directly obtained from the decomposed products in the food by means of the acid in the stomach. On the other hand, when the gases are not directly derived from decomposed products in the swallowed food, they are formed by putrefaction of the albuminous contents of the intestinal tract. It seems probable that in the majority of these cases, the inflammable gas (marsh gas) is derived from the intestine where putrefactive decomposition of proteids readily occurs. This appears to have been the condition in M'Naught's case already quoted. Taking all the facts together—especially the greater ease with which putrefaction occurs in the intestine than in the stomach—it may be concluded as probable, as Hoppe-Seyler considers, that the marsh gas eructated in some cases of disease is formed in the intestine and not in the stomach.

As examples of the composition of eructated gases the following analyses[1] may be given:—

	Popoff.	Schultzen.	Ewald and Rupstein.	Frerichs.
Carbonic acid, CO_2 . .	12·82	26·56	17·40	20·57
Hydrogen, H_2 . . .	32·32	32·30	21·52	20·57
Nitrogen, N_2	46·96	33·44	46·44	41·38
Oxygen, O_2	7·90	7·36	11·91	6·52
Marsh gas, CH_4 . . .	—	0·34	2·71	10·75

The Digestive Activity of the Gastric Juice in Disease and the Variations in the Mechanical Movements of the Stomach.—The increase or diminution in the amount of hydrochloric acid with or without the presence of organic acids and the evolution of gas is only one part of the changes which may occur in the stomach in disease. There are also changes in the amount of pepsin secreted, in the movements, and in the absorptive activity of the organ. The chief changes occur in the amount of pepsin secreted and in the mechanical movements of the organ. The digestion and manipulation of the mixed food in the stomach does not depend on any one function of the organ; besides the fermentative changes produced by the pepsin-hydrochloric acid of the gastric juice, efficient digestion cannot occur unless the normal movements of the organ are continuous throughout the whole period of digestion; for, however powerful the gastric juice may be, the food must be intimately mixed for the chemical process to be accomplished within a certain time, and it must after the process is ended be completely propelled through the pylorus.

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[1] See Halliburton's "Chemical Physiology and Pathology," p. 651; and Gamgee's "Physiological Chemistry," vol. ii. p. 174; Popoff, *Berl. klin. Wchnschr.*, 1870, Nos. 38 and 40; Schultzen, *Arch. f. Anat. u. Physiol.*, 1864, p. 591; Ewald and Rupstein, *Arch. f. Anat. u. Physiol.*, 1874, p. 217.

3. *The Digestive Activity of the Gastric Juice in Disease.*—No quantitative estimation of pepsin as a substance that is of any practical value can be made; the amount contained in a particular liquid obtained from the stomach is gauged by the rapidity of its action on albuminous substances (*e.g.* egg-albumin) at a certain temperature (35° to 40° C.) in the presence of 0·2 per cent hydrochloric acid.

The digestive activity of the gastric juice may be tested in a particular individual in two ways:—

(*a*) Either by giving a test-meal and ascertaining its digestibility by an examination of the stomach contents which are removed some time afterwards;

(*b*) Or during fasting, by exciting the stomach to secrete in various ways and removing the stomach contents in ten to fifteen minutes (see Chapter V.).

Leube used in his investigation of the digestive activity of the stomach a test-meal of soup, a large beef-steak, and a small white roll. After seven hours, during which no other food was taken, the stomach was washed out with a minimum of water and the washings tested. Undigested residue was obtained if the digestion was not complete, but in cases of healthy people, with one or two exceptions, the digestion of this meal was complete in seven hours. He tested the secretory activity of the stomach by injecting into the stomach 100 cc. of ice-cold water, removing it in ten minutes by adding 200 cc. of water. The digestive activity of this liquid, made to the proper degree of acidity by means of hydrochloric acid, was tested by placing some coagulated egg-albumin into it and allowing it to stand at the temperature of the body in an incubator. In all cases the same amount of egg-albumin was used so that a comparison could be made of the digestive activity of the juice in the cases observed. Without discussing at this point the value of these methods, the results obtained by Leube and by other observers using similar methods may be described.

In five healthy individuals, Leube found that this test-meal was completely digested in seven hours, and that the gastric juice removed from the stomach digested the egg-albumin in from one to two hours. In two apparently healthy individuals this was, however, not the case, viz. the test-meal was not digested in seven hours, and the digestive activity of the gastric juice was very small.

Fourteen cases of "nervous dyspepsia" were examined: in twelve the test-meal was digested in seven hours, in two it was undigested. In these last, the activity of the gastric juice, tested separately, was greatly diminished. Out of eight cases in which the amount of hydrochloric acid was tested, five showed a deficiency. According to these results, therefore, in the majority of cases of "nervous dyspepsia," the chemical digestive power of the stomach is not diminished to any extent.

In nine cases of "chronic dyspepsia" the test-meal was undigested in seven hours, and the activity of the gastric juice was greatly diminished, the diminution varying in individual cases. In six cases of chronic dyspepsia with dilatation of the stomach a similar result was obtained; also in three cases of carcinoma ventriculi.

The non-digestion of the test-meal and the insufficiency in the digestive activity of the gastric juice are most marked in cases of acute catarrh of the stomach. During the acute attack the test-meal was undigested; during convalescence it was digested, and the gastric juice secreted was very active. In atrophic stomach catarrh, Jaworski found in some cases a diminution of pepsin, and in some cases of pulmonary tuberculosis Brieger found that there was a general insufficiency of the secretion of gastric juice.

Korczynski and Jaworski investigating twenty-four cases of ulcer of the stomach by means of a test-meal of egg-albumin found that the digestive activity was increased.

Riegel, and Gluzinski and Jaworski found in some patients not suffering from ulcer, hypersecretion and increased activity of the gastric juice.

On the whole, the conclusion is that in the majority of cases of functional disorder of the stomach, there is but little diminution in the amount of pepsin secreted; that in fact the secretion of hydrochloric acid is more sensitive to disease than that of pepsin. In organic disease of the mucous membrane, such as occurs in inflammation, degeneration, and in cancer, the secretion of pepsin is, however, profoundly affected.

Pepsinogen.—It was previously stated that pepsinogen was the precursor of pepsin, that it was secreted as such by the glands and transformed into pepsin by means of the hydrochloric acid. In disease, therefore, it is quite possible that pepsinogen may be secreted and not become transformed into pepsin, owing to the deficiency or absence of hydrochloric acid. This question, however, is very difficult to investigate, inasmuch as the separation of pepsin and pepsinogen requires very careful procedure: the chief differences between the two substances being that pepsin is destroyed by sodium carbonate, and that pepsinogen is precipitated and destroyed by a current of carbonic acid passed through its solution (Langley). It is easy to prove in a liquid removed from the stomach that both pepsin and pepsinogen are absent, but very difficult to show that one or other or both are present. Pepsinogen is more resistant than pepsin; and, according to Boas,[1] it may be found in acute catarrh and in cancer of the stomach, when pepsin is absent. Further investigation is needed, however, before the question of the occurrence of pepsinogen in disease can be discussed: all that can be said is that it is probable, that in some cases where there is a deficiency of secretion of hydrochloric acid, pepsinogen and not pepsin may be found in the stomach contents. In neutral or very slightly acid vomit in cases of catarrh, the liquid when made acid may show great digestive activity (Chapter V. p. 141); but this is no proof that pepsinogen is present and transformed by the addition of the acid, as pepsin in a similar liquid would act in the same manner.

Curdling Ferment (*Rennet*). — The presence of this in disease has been investigated by Boas,[2] and Boas and Tozebinski. The curdling ferment has a precursor which bears the same relation to it as pepsinogen does to pepsin, and is transformed into the ferment by the action of acid and of warmth. These observers found that the ferment was present in varying amounts in different conditions of the stomach. It might be normal in amount, even in the presence of a varying amount of hydrochloric acid: as in "neuroses" of the stomach. It might be greatly diminished; as in serious organic disease—catarrh, carcinoma, or degeneration of the mucous membrane.

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[1] "Diagnostik u. Therapie der Magenkrankheiten," 3rd ed. part i. p. 22.

[2] *Op. cit.* part i. p. 190.

4. *Variations in the Movements of the Stomach* (see also Chapters V. and XIV.).—Unless the normal movements of the stomach go on during the whole time the food is in the organ, digestion, even in the presence of a normal secretion of gastric juice, is not only not complete, since the digesting food is not intimately mixed, but the stomach is never completely emptied since the organ is incapable of propelling the last portion of its contents into the duodenum. These two results are of great importance in the pathology of indigestion of food; undigested or imperfectly digested food is propelled in the one case into the duodenum, and in the other the stomach always contains some food which may undergo bacterial fermentation.

Variations in the movements of the stomach are of two kinds; either in the form of increased or in that of diminished movement, the latter being by far the most common change.

Increased movements of the stomach occur both in functional and in organic diseases of the organ. Thus they are a feature of some cases of lienteric diarrhœa, and of gastro-intestinal disorder, especially in children. Shortly after food has been taken, there is rapid peristalsis starting from the stomach and leading to the passage of a liquid stool, which in children at any rate often contains undigested food. Such a condition must be described as one of increased reflex excitability. An increase of reflex excitability of the movements is observed in other cases of functional disease, especially in cases of gastric irritation and of neurosis.

Thus spasm may occur, either of the orifices (*e.g.* of the cardia) or of the whole stomach. The latter is seen when the stomach contains very irritating food; the former is observed in neurotic individuals, and occurs in relation to food.

In organic disease increased movements are observed when there is pyloric obstruction, the peristaltic movements being increased to overcome the obstruction (see "Dilatation of the Stomach," Chapter XIV.).

In most cases of functional and organic disease, the movements of the organ are diminished, and this condition is called *atony* or *myasthenia* (Boas).

In cases of dilatation of the organ, the movements are diminished; dilatation is, so to speak, the expression of a diminution in the movements. But the movements may be diminished even when no dilatation of the organ can be detected.

Thus in chronic gastric catarrh, in "nervous dyspepsia," and in dyspepsia in pulmonary tuberculosis, the movements have been found diminished (Klemperer, Herzog). In ulcer, Korczynski and Jaworski also found a deficiency of movement; and a similar observation was made by Klemperer in cancer of the pylorus.

The results of these direct observations confirm what has been noticed from clinical observation of these cases. Deficiency of movement plays as great a part in the production of disordered digestion as deficiency of secretion of gastric juice. It is the direct result, as has been pointed out, of irregularity of diet, of the partaking of too large meals, and of meals with a large quantity of indigestible matter or of food accessories, whereby the organ has more than it can digest or manipulate, and is active for too long a period. The over-worked organ at last ceases to respond to stimulation, and not only the secretion but the movements diminish and finally cease. Indeed, a large meal may in some cases induce an actual paralysis of the stomach wall, with the production of sudden and acute dilatation, threatening life or even causing death (see "Dilatation of the Stomach," Chapter XIV.).

This is a rare event, but a deficiency of movement of the stomach from whatever cause is greatly influenced by the character of the food taken, so that large meals, or meals composed of improper and indigestible food, aggravate the condition.

Another cause of deficient movement of the stomach is to be ascribed not directly to the food taken, but to some general condition of the body which reacts on the organ. Although not actually proved by experiment, it may be concluded as probable and explanatory of some of the symptoms observed, that in fever, anæmia, and in chronic wasting diseases, such as cancer and pulmonary tuberculosis, there is a deficiency in the movements of the stomach. The state of fever tends to diminish muscular movements, both voluntary and involuntary; and an anæmic condition, whether as regards simply diminution of the hæmoglobin or of the red corpuscles, produces the same effect.

The nervous system plays a great part in producing a deficiency of movement of the stomach, a deficiency which may be the commencement of dilatation of the organ of varying degree. This is observed not only in the cases classed as nervous dyspepsia, but in very many cases of functional disorder which develop in consequence of the mode of life of the individual, or which are associated with certain conditions of nervous irritability, especially in women (Chapter VII.). In organic disease of the stomach the movements are often greatly diminished: in ulcer and cancer, and in extensive degeneration of the mucous membrane of the stomach.

There may be an actual mechanical hampering of the movements from external causes, such as pressure on the duodenum near the pylorus, adhesions round the organ near the pylorus or near the cardia, in some instances due to old peritonitis, in others to a healed pleurisy with retraction, especially when left-sided.

The most important final result of deficiency in the motor activity is dilatation of the organ with or without bacterial fermentation of the food, this dilatation being, as has been previously remarked, an expression of the deficiency of movement. Another result is flaccidity and patency of the pylorus, which allows the fluids and gases of the small intestine to enter the stomach.

5. *Absorption by the Stomach in Disease.*—It was stated (p. 26) that normally the stomach did not absorb the water drunk, but that it did absorb salts to some extent, and to a greater extent alcohol and sugars (lactose, maltose,

glucose, and cane-sugar after transformation). Peptones are absorbed to a slight extent, and during the process disappear as such, being retransformed by the mucous membrane into the proteids of the blood. Reasons have been given which render it probable that most of the digested proteids pass into the duodenum, to be further acted upon by the pancreatic juice and the bile, so that the non-absorption of peptones by the stomach must be considered as a subsidiary matter in disease.

As regards the water drunk, there is much evidence to show that very little is absorbed by the diseased stomach. Thus, in a dilated stomach, the contents of the stomach contain a large quantity of water, even many hours after food; also, when the stomach is washed out, and liquid food again introduced, this disappears only in small quantity after the lapse of hours. In such cases of disease there is therefore very little, if any, absorption of water by the stomach: and the same may be said of cases where there is a deficiency of movement without dilatation. The absorption by the stomach depends on the proper continuance of the blood-supply to the organ, and to some extent on the degree of concentration of the stomach contents. In disease, absorption may be influenced by two conditions. The first is the secretion by the stomach of a large excess of mucus (as in catarrh), which lines the mucous membrane, and acts as a mechanical barrier to absorption. The second is the state of congestion of the organ. In both mechanical and active congestion, the tendency is to stasis of the blood-stream, in the first case due to an obstruction of the venous circulation, in the second to an effect on the arteries, leading to stoppage of the circulation. Both these conditions are very different therefore from the increased blood-supply of the stomach during digestion, for in this case, as in the first stage of active congestion, there is an increased flow of blood through the vessels with no tendency to stasis. This increased flow of blood, or "blushing of the stomach," is the great aid to absorption.

The non-absorption of the sugars by the stomach in cases where there is delay of food in the organ, is an important factor in the production of symptoms, since it is the sugars which undergo bacterial fermentation leading to the formation of gases and of organic acids.

There is as yet no accurate method by which the absorption of sugars and of peptones by the stomach in diseased conditions can be studied. Attempts have been made to study the absorptive activity of the stomach by means of iodide of potassium, a rapidly diffusible salt which, when taken into the stomach, is excreted in a few minutes in the saliva and the urine. The results which have been obtained are not without interest, but must be considered as referring chiefly to the particular salt used, since all substances are not absorbed in the same way. The sugars and albumoses have to be transformed (p. 25) before they enter the blood-stream; iodide of potassium is absorbed as such, and is excreted in the saliva and urine as a simple iodide. It may readily be detected in these liquids by adding to them a little fuming nitric acid, which sets free the iodine. The presence of this is indicated in small amounts by turning starch paper a violet-red, or in large amounts by turning it blue. The iodine may also be separated by adding chloroform and shaking; chloroform dissolves it, and sinks to the bottom of the mixture as a red liquid.

Zweifel,[1] in using this method for testing the absorptive activity of the stomach, gave about 3 grains (0·2 gramme) of iodide of potassium in a gelatin capsule; 3½ oz. of water (100 cc.) being drunk at the same time. He found that on an average, in twenty different healthy individuals, the iodide could be detected in the saliva in from eight to ten minutes. It appeared somewhat later in the urine. The absorption time does not vary much in the same individual, except when the stomach is full. In this case the absorption time is not only prolonged, but is very irregular in the same individual. This prolongation of absorption in a full stomach is probably due not only to the dilution of the iodide by the stomach contents, but to the fact that the salivary glands are not so active some time after a meal as in the fasting condition. The results of these experiments are indeed somewhat minimised from our present point of view by the fact that it is not only the absorptive activity of the stomach that is being investigated, but also the excretory activity of the salivary glands. Moreover, it is not at all certain that the gelatin capsule with the water swallowed does remain in the empty stomach. A small amount of gastric juice would be secreted, which would readily dissolve the gelatin, setting free the iodide; but it is quite possible, and indeed probable, from what we now know of the absorption of water in the stomach, that most of the liquid containing the iodide passes rapidly through the pylorus. The results of the experiments may therefore express not only the absorptive activity of the stomach and the excretory activity of the salivary glands, but also the absorptive activity of the small intestine.

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[1] "Die Resorptionsverhältnisse der menschlichen Magenschleimhaut," *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxxix. p. 349, 1886. In this paper an abstract of the previous work on the subject is given.

Bearing these criticisms in mind, Zweifel's conclusions, which more or less confirm those of previous observers, who used a similar method, may be considered.

His conclusions are that in nearly all diseases of the stomach there is a tendency to prolongation of the absorption; greatest in dilatation and in carcinoma of the stomach, least in chronic catarrh, and only slight in ulcer. In the early stages of ulcer, however, the absorption time may be greatly prolonged. Moreover, if in the fasting stomach, the absorption time of iodide of potassium is greater than twenty minutes, there is a probability, if ulcer be excluded, that the case is one of stomach dilatation, or carcinoma pylori. No differential diagnosis by this method can be made between ulcer and chronic catarrh of the stomach; and both in ulcer and in dilatation the absorptive activity of the organ may become normal under treatment. Zweifel does not consider that the results of this method of testing the absorptive activity of the stomach are of great importance in the diagnosis in this particular instance; but they may be of help in addition to the clinical features of the case, and to the chemical examination of the digestive activity.

Several cases of fever were examined by him: pulmonary tuberculosis, pneumonia, and typhoid fever, in some of which the temperature was rising and in others falling. The absorption time was prolonged as compared with an afebrile individual, and bore no relation to the degree of fever. Granting that the method used is a test of the absorptive activity of the stomach, it would appear probable that the prolongation of the absorption time is dependent on altered conditions of circulation in the stomach, that is to an irregularity in the normal response to stimulus of the blood vessels of the organ.

Summary.—The functions of the stomach which are mainly altered in disease are the chemical processes of digestion, dependent on the secretion of an adequate quantity of hydrochloric acid and of pepsin, and the movements of the organ. Absorption by the diseased stomach is a condition so little known that no adequate account can be given of it. In disease, the functions of the organ may be either increased or diminished. The gastric juice is secreted in response to the stimulus of the food even when it is in the mouth, but much more so when it is in the stomach. The direct exciter of the movements of the stomach is the presence of food in the organ. If the stimulus is increased in an individual case, these functions will up to a certain point also be increased. An increased stimulus would be such as an increased quantity of food and an increase in the condiments and of the other food accessories. The food accessories, especially alcoholic drink and tea, are irritants in excess. An increased stimulus leading to increased function of an organ soon becomes an irritant leading to diminished function. This is the case with food, for if this is taken in repeatedly large quantities, or with a large amount of food accessories, the functions of the stomach are diminished and indigestion of food results. Two stages may thus be distinguished, the early stage of increased function, and the later stage of diminished function. In disease, both functional and organic, of the stomach, it is usually the second stage which comes under notice; the first stage being usually of short duration. The various degrees, however, in which the secretion of gastric juice and the movements of the organ are affected in disease of the stomach are of prime importance in diagnosis and treatment; and in each individual case it is the object of the physician to discover how far these functions are affected.

This is by no means an easy task; but in many cases it is readily determined which function is the most affected. What is known on the subject may be conveniently and shortly stated in the following table:—

	Pepsin.	HCl.	Movements.	Absorption.
1. Gastric irritation due to irregularities in diet: early stage	Increased.	Increased.	Increased or diminished.	Doubtful.
Do. late stage	Diminished.	Diminished.	Diminished.	Slightly diminished.
2. Gastric insufficiency	Diminished.	Diminished.	Diminished.	Slightly diminished.
3. Chronic catarrh 4. Dilatation, simple	Affected in prolonged cases.	Diminished.	Diminished.	Slightly diminished.
5. Acute catarrh 6. Atrophy of mucous membrane	Greatly diminished or absent.	Greatly diminished or absent.	Greatly diminished.	Diminished.
7. Ulcus ventriculi	Normal or increased.	Normal or increased.	Diminished.	Diminished.
8. Carcinoma ventriculi	Normal or diminished.	Diminished or absent.	Diminished.	Diminished.
9. Fever	Diminished.	Diminished.	Diminished.	Diminished.
10. Anæmia	Diminished.	Diminished.	Diminished.	Doubtful.
11. "Nervous" dyspepsia	Normal.	Normal or variable.	Normal or diminished, variable.	Normal.

This table does not show all the conditions present in each particular disease, and the variations found will be more fully discussed when the symptoms are considered. It may be well, however, to again draw attention to the following points:—

1. That "chronic dyspepsia" is a condition due to three several changes in the stomach; two of which are functional, viz. gastric irritation and gastric insufficiency, and one of which is due to organic disease, chronic catarrh.

2. In these conditions, the chief change in the stomach lies in alterations in the secretion of the hydrochloric acid and in the movements of the organ: the secretion of pepsin being affected to a less extent.

3. That a profound effect on all the functions of the organ is observed in acute catarrh and in degeneration of the mucous membrane.

4. That in ulcer of the stomach, the secretion of the gastric juice may not only be normal, but actually increased in quantity.

5. That in cancer of the stomach, there is a general diminution in the quantity of pepsin and hydrochloric acid secreted and in the movements of the organ.

In all these cases, it may be said that the functional activity of the stomach depends on the duration of the diseased condition, but even in the last stages of disease the stomach still preserves some digestive activity and some degree of movement. With acute catarrh, there is a great diminution and in some cases a cessation of functional activity of the stomach; this stage lasting only a short time. But in atrophy of the mucous membrane, whether primarily due to the action of corrosive and irritant poisons or to an acute or subacute inflammation becoming chronic, the functional activity of the stomach is permanently affected.

6. *The Process of Digestion of the Mixed Food in the Disordered Stomach.*—During normal digestion, as has been already fully described, the following events take place in the stomach.

On the entrance of food into the organ, the secretion of gastric juice and the movements at once commence, and the organ assumes a pink colour owing to an increase of flow of blood to it. During the process of digestion, which with a moderately large meat meal lasts from five to seven hours, the hydrochloric acid of the gastric juice gradually increases to a maximum in from one to three or four hours, and then gradually decreases. The movements also, at first slight, gradually increase in intensity. During digestion the organ is practically a closed bag, and although, at intervals, a small amount of the liquid food is discharged through the pylorus, yet the greater part of it is not discharged until the end of digestion, but little of the water being absorbed by the stomach. At the end of digestion, the stomach remains quiescent, but in a contracted state, and contains a small amount of neutral or slightly alkaline liquid with a few flakes of mucus. When the next meal is taken, the processes are repeated, and in all cases without any sensation on the part of the individual except one of repletion and contentment.

In disordered digestion, whether there is organic disease of the stomach or not, the conditions are very different. Digestion in such cases has to be carried on under four different conditions:—

1. Where there is an increased secretion of gastric juice.
2. Where there is a diminished functional activity of the stomach.
3. In the presence of bacterial fermentation of the stomach contents.
4. In varying conditions of irritability and of congestion of the stomach walls.

1. When there is hypersecretion of gastric juice, the chemical processes of digestion are well performed, the mixed food being rapidly digested, and the stomach in most cases rapidly emptied. In such cases, which may be only the early stage of diminished functional activity of the stomach, there are symptoms which are referable to the high degree of acidity of the stomach contents. Diminished motor activity may, however, be associated with hypersecretion, and in this case there is delay of food in the organ with prolonged distress.

2. When the functions of the stomach are diminished, the chief effect on digestion is that the process is delayed. Such diminution of function occurs not only in functional disorder of the stomach, but also in cases of organic disease such as long-standing cases of ulcer and in cancer. The general result is delay of food in the organ, due not so much to the absence of pepsin as to the diminution in the amount of hydrochloric acid and in the movements. When a mixed meal is taken in such conditions, there is slow manipulation of the food and slow digestion owing to the insufficiency of hydrochloric acid present, which never rises to the normal of 0·2 per cent. Instead of taking five to seven hours

to digest, the food remains in the organ for over seven hours, and the stomach is not empty when another meal is taken. At this time, or before this, the chemical processes of digestion have actually ceased; no more pepsin and hydrochloric acid is secreted, and the hydrochloric acid may gradually disappear by absorption. The liquid that remains is in part expelled through the pylorus, but owing to the weakness of the muscles of the organs, it is not all propelled, and some remains. The stomach, therefore, is never quite empty, and the characters of its contents in over seven hours after a mixed meal vary in different cases, according to the functional activity of the organ. The results of the examination of the stomach contents are more fully discussed in Chapter V.; it is sufficient here to say that in such conditions they are divisible into three classes:—

(*a*) Those very acid, with a large amount of undigested residue of food, and with peptones in solution.
(*b*) Those slightly acid or neutral, with very little, if any, undigested food; peptones being present, and often pepsin.
(*c*) Those neutral or alkaline with a sweetish taste, and containing peptones, and in some cases bile and liquid from the duodenum.

The delay of food and of its results, as just indicated, are greatly increased when there is dilatation of the stomach. In such cases, whether simple or obstructive, the stomach always contains a liquid, sometimes in large quantities, which cannot pass through the pylorus, either because this is obstructed or because the muscles are unable to propel it forward.

3. The presence of bacterial fermentation of the food in the stomach is of great importance in considering the digestion of food by the disordered stomach. The two conditions which favour it are the diminution in the amount of hydrochloric acid secreted and the delay of food in the organ owing to the weakness of its muscular walls. The products of bacterial fermentation which hinder digestion in the stomach are derived from the carbohydrates, and are the organic acids—

lactic, butyric, and acetic acids. There is but little known of the products of the bacterial fermentation of proteids in the stomach, and even if these were present, it is unlikely that they would influence the chemical processes of digestion.

An increase in the organic acidity of the stomach contents, whether due to lactic, butyric, or acetic acids, is inimical to the rapid digestion of the mixed food by the gastric juice. The chemical processes of digestion proceed much more slowly in the presence of these acids than in that of hydrochloric acid (p. 34); and, moreover, when there is an excess of these acids, as in advanced bacterial fermentation, the action of the pepsin is actually inhibited, even though hydrochloric acid may be present.

The conditions of bacterial fermentation are those of delay of food in the stomach, and in such cases the stomach is never empty, but contains after digestion has ceased an amount of liquid varying in quantity but always very acid. When, therefore, the next meal is taken it mixes with this acid liquid and its degree of acidity at once rises before the secretion of hydrochloric acid. The result is, as has been shown by actual observation, that the secretion of hydrochloric acid is delayed, and thus digestion is still further embarrassed. The permanent acidity of the stomach due to these organic acids, therefore, actually leads to a diminution in the secretion of the hydrochloric acid.

Another aspect of the influence of organic acidity on digestion may here be mentioned, although there may be no bacterial fermentation present in the particular cases. In normal digestion, a relatively large quantity of certain vegetable acids, such as acetic, citric, tartaric, and malic acids, may be taken without any discomfort; although their continued use in large quantities leads to disordered digestion. Moreover, from neutral fats fatty acids may be liberated in the stomach, and from the ordinary fats eaten, which always contain free acid, this is set free. In cases of disordered digestion, with a diminution of functional activity of the stomach, the presence of these organic acids in the food by increasing the organic acidity of the stomach contents delays the chemical processes of digestion, and they act, like the organic acids of bacterial fermentation, as irritants. They have other effects, chiefly on intestinal digestion, which are considered on p. 117.

4. Varying conditions of irritability and congestion of the stomach walls have an important effect on the digestion of food in the stomach. The gastric juice is secreted in response to the stimulus of the food in the organ: the movements result from the same stimulus, and both these functions are associated with an increased blood-supply, which is also secondary to the stimulus of food.

It is known from the direct observation of Dr. Beaumont in the case of Alexis St. Martin that the stomach may be in such a condition that a stimulus applied to the mucous membrane produces no secretion of gastric juice as it normally does, and no movement, or only a slight one. This condition of irresponsiveness of the stomach, although not so complete as in the case quoted, is no doubt common in cases of disordered digestion. An organ in response to increased stimuli will perform its functions normally up to a certain point; but in time the increased stimulation leads to diminution of function. This is true of all normal tissues. It is of great importance as applied to the stomach, for the irresponsiveness, great and little, of the organ to the normal stimulation of the food leads directly to a diminished secretion of gastric juice, to diminished movements of the organ and to altered conditions of blood-supply, on which both the continued secretion of juice and the continuous movements are dependent. That this condition of things is present in cases of disordered digestion is not deduced from the results of direct observation; this is not possible. But it is a fair deduction from the facts known regarding the functions of the stomach in health and disease. In indigestion of food directly due to the breach of dietetic rules, it no doubt plays an important part in leading to the continuance of disordered digestion; for in such cases we have a typical example of an organ overloaded with food which may be harmful not only from its bulk, but from its indigestibility, its acidity, and its admixture with an excess of condiments and alcoholic food accessories.

7. *Relation of Indigestion of Food in the Stomach to other Changes in the Alimentary Tract.*—The normal relations between the digestive processes in the stomach to those occurring in other parts of the alimentary tract (the mouth and intestines) have already been discussed (p. 36). When there is disordered digestion in the stomach there may be changes both in salivary and in intestinal digestion; and although very little is accurately known of the subject, yet certain facts have been observed which are of pathological importance. The chief points for consideration arise in those cases: (1) where there is an excess of acidity of the stomach contents during digestion, and (2) where there is a diminution in the acidity. In the first class the excess of acidity may be due either to hydrochloric acid or to hydrochloric acid and organic acids (lactic, butyric, acetic, citric, tartaric, etc.); in the second class there is a diminution in the secretion of hydrochloric acid.

With diminished acidity of the gastric contents, there is imperfect digestion of proteids in the stomach, but a rapid and effective digestion in the small intestine, the digestion of starch being also very active (Boas).[1] But in this case, owing to the diminution of acidity in the stomach, there is not sufficient hydrochloric acid to decompose the bile-salts, so that the bile acids are not set free. The contents of the small intestine are thus liable to undergo bacterial decomposition; the chief change probably being the bacterial decomposition of the carbohydrates, whereby lactic and butyric acids are formed. With hyperacidity of the stomach contents, due to hydrochloric acid, there is rapid and efficient digestion in the stomach, but when the acid stomach contents enter the duodenum, the intestinal contents remain acid for some time, the digestion of the proteids progresses but the digestion of fat and of starch is delayed, at least for a time.

In some cases of hyperacidity which come under the heading of "acid dyspepsia" (gastric irritation), there is, at any rate, in some individuals, an increased alkalinity of the saliva, which may be equal to 0·125 to 0·15 per cent HCl. This would be an alkalinity of about 0·18 to 0·2 per cent sodium carbonate ($Na_2\,CO_3$); which is much greater than the average normal found by Chittenden and Smith (0·097 per cent, $Na_2\,CO_3$). This increased alkalinity of the saliva may be looked upon as partly a reflex effect from the action of the hyperacid contents of the stomach, but also as an increased excretion of carbonates by the saliva. If an excess of sodium carbonate is injected into the blood, a large excretion of very alkaline saliva takes place; and in the cases under consideration of an increased secretion of alkaline saliva, the excess of carbonates in the blood might come from the hyperacid stomach contents, from which the vegetable acids (citric, tartaric, lactic, acetic) are absorbed and changed into carbonates in the system. Part of the absorbed carbonate is excreted in the urine, but part also in the saliva. The increased secretion of saliva probably has a salutary effect, inasmuch as when swallowed it helps to neutralise the acids of the stomach and thus to diminish the hyperacidity of the contents.

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[1] *Ztschr. f. klin. Med.*, Berlin, Bd. xvii. p. 155.

More important effects probably result from the entrance into the duodenum of hyperacid chyme, when the hyperacidity is due to hydrochloric acid and to an excess of organic acids. Part of the acidity will be neutralised by the bile and pancreatic juice; but not all, so that the continued acidity of the intestinal contents will in the early stage of digestion delay the digestion of fat and of the carbohydrates. The presence of free organic acids in the duodenal contents has probably an effect other than that due to this degree of acidity, viz. that in small quantities they stop the digestion of starch by the pancreatic diastase. V. Hofmeister[1] found that as small a proportion as 0·04 per cent of lactic acid delayed the digestion of starch, while 0·05 per cent completely stopped it; while with acetic acid, 0·05 to 0·6 per cent diminished and 0·08 per cent stopped digestion. So small a percentage of these acids would not uncommonly exist in the duodenum in cases of high organic acidity of the stomach contents in the early stages of intestinal digestion, although later the acidity would be neutralised. Another effect of the presence of organic acids in the intestine, but only when they are found in large quantities, is the production of diarrhœa. Experimentally,

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[1] Abst. in Maly's *Jahresbericht*, 1889, Bd. xx. p. 266.

Bokai[1] has shown that lactic, tartaric, butyric, formic, acetic, propionic, caprylic, and caproic acids act in this way. Diarrhœa alternating with constipation is one of the results of functional disorder of the stomach (gastric irritation), and is in part due no doubt to the presence of organic acids in the intestine. These acids, however, do not all come directly from the stomach, some fatty acids are formed by the pancreatic juice by the splitting up of neutral fats into glycerine and fatty acids, others come from the stomach contents, and others again are formed in the small intestine, especially in the lower part, by the bacterial fermentation of carbohydrates. For a further discussion of the condition of the intestines, see Chapter X. p. 307.

8. *Condition of the Urine in Disease of the Stomach.*—In physiological conditions, the urine varies somewhat according to the food taken. After a mixed meal, composed of proteids, carbohydrates, fats, and salts, the acidity of the urine (which is due to acid sodium phosphate) diminishes, and at a varying interval from ten to five hours, becomes alkaline; it then regains its acidity. The alkalinity is due to the liberation of bases during the secretion of hydrochloric acid in digestion. The urine passed after fasting is acid; and it is acid or only slightly alkaline after a meal consisting chiefly of meat. The internal administration of acids and prolonged muscular exertion also render the urine acid.[2]

From the point of view of stomach disorder, it is important to note that there is a relation between the secretion of hydrochloric acid and the reaction of the urine. The urine in stomach disease is affected by the amount of hydrochloric acid secreted by the stomach, by the presence of bacterial fermentation of the food and of dilatation of the stomach, and by the presence of organic disease.

The *hypersecretion of hydrochloric acid* tends to produce alkalinity of the urine; as a rule, a diminished excretion of chlorides and an increased excretion of phosphates. A diminished secretion of hydrochloric acid does not affect the reaction of urine to any great extent, even in the period following a meal. The amount of chlorides present varies, but does not show great variations from the normal; phosphates may in these cases be increased.[1]

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[1] Abst. in Maly's *Jahresbericht*, 1889, Bd. xv. p. 298.

[2] See Halliburton, "Chemical Physiology," p. 714.

In *dilatation of the stomach* the urine shows the same condition as in diminished secretion of acid, but it is passed in greatly diminished quantity. When there is advanced bacterial fermentation in addition to the changes associated with dilatation of the organ, the urine has been found to contain an excess of the ethereal sulphates which are derived from putrefactive processes occurring in the gastro-intestinal tract. It is more probable that the ethereal sulphates are formed in the intestine than in the stomach where putrefactive processes are rare. An increase in the amount of indican has also been found in these cases, but it, like the other ethereal sulphates, is more commonly derived from putrefactive changes in the intestinal contents than in those of the stomach.

In *organic disease of the stomach*, besides the conditions in the urine mentioned above, as associated with the changes in the secretion, there may be albumosuria (peptonuria). This also occurs in dilatation of the stomach (simple or due to carcinoma) and in chronic ulcer. In cancer and in ulcer of long-standing there is an increase in the excretion of urea.

In some acute and obstructive affections of the stomach, acetone and diacetic acid have been found in the urine, which gives the blood-red coloration with ferric chloride solution. These conditions are severe forms of dilatation, such as occur in stenosis of the duodenum, and in carcinoma (Boas); and in some other acute and chronic stomach affections.

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[1] For the literature on this subject see Boas, *op. cit.* part i. p. 232.

CHAPTER V.

METHODS OF EXAMINATION OF THE FUNCTIONS OF THE STOMACH IN DISEASE.

It is evident from the facts brought forward in the last chapter that the examination of the contents of the stomach during digestion is of great service in the diagnosis and treatment of disordered digestion. The examination of vomited matters will in many instances indicate the conditions of digestion: and when the character of the previous meal is known and the period elapsing before vomiting, the examination is nearly the same as giving a test-meal to the patient and withdrawing the stomach contents after a certain interval. The examination of vomited matters, and of liquids withdrawn from the stomach after a test-meal or otherwise, is conducted on the same lines.

In some cases it is advisable to determine the digestive activity of the gastric juice, and the mechanical power and absorption activity of the stomach.

I.—Examination of Vomit and of Liquids Removed from the Stomach.

These liquids must be examined for:—

1. The presence of undigested food.
2. The presence of micro-organisms.
3. The presence of mucus, pus, and blood.
4 The total degree of acidity.
5. The character of the free acids present: hydrochloric, lactic, butyric, acetic.

6. The presence of the products of digestion: syntonin, albumoses, peptone.
7. The presence of pepsin and the curdling ferment.

The examination of the stomach contents is of great importance, and can, to a large extent, be performed by such simple methods that it may be conducted with as much ease as the examination of the urine. The results obtained often throw much light on the process of digestion in the disordered stomach; in the case both of vomited matters and of liquids removed from the organ at a definite period after a test-meal. The methods of examination which will now be considered are those most easy of application, those which ought to be applied in the examination of the stomach contents.

1. But little need be said of the examination of the undigested residue of the food. In some cases the examination is important as showing the character of the food taken, such as the skins of fruit, coagula of milk, undigested muscle fibres, starch, etc. It may be important to determine whether in a given case, the food, *e.g.* muscle fibre, is actually in the process of disintegration, for the diagnosis between regurgitation of food from the œsophagus and the vomiting of food; in the former case the muscle fibre is not in a state of partial solution. Starch is recognised by the blue colour it gives with iodine, and sugar by means of the reduction of Fehling's solution.

2. A bacteriological examination is important, and, clinically, a microscopical one is sufficient. Cover-glass preparations stained with methylene-blue or unstained may show the presence of various forms of bacteria—bacilli (long and short) and cocci—or the presence of torula or sarcina. The presence of bacteria in the recently evacuated stomach contents discoverable by a microscopical examination is a sign of fermentation in the stomach, which is also demonstrated by a chemical examination of the liquid.

Yeast (torula, saccharomyces) and sarcina are discovered in the stomach contents only in advanced cases of fermentation, and almost invariably when the organ is greatly dilated. They are readily recognised by their characteristic forms (see

Chapter III., Figs. 15 and 16). Sarcina are best studied unstained. They are difficult to stain well, as they so readily take up the aniline dyes, and then look like blackish patches: they may, however, be well stained by drying a drop of the stomach contents on a cover-glass and placing in a very weak solution of gentian-violet (so weak as to be nearly transparent) for a few minutes; washing in water, drying, and mounting in Canada balsam. Yeast is best studied when faintly stained in methylene-blue solution, viz. for half a minute in a 2 per cent solution, afterwards washing well in water.

3. *The Presence of Mucus, Pus, and Blood.*—The *presence of mucus* in the stomach contents is evident to the naked eye: its stringy and tenacious characters are sufficient for a diagnosis when in large quantity. A chemical diagnosis of the substance it contains, viz. mucin, is made by placing some of the mucus in some cold water and gently washing it: pouring off the water, and adding a little liquor potassæ or baryta water in which mucin is soluble and forms a solution from which it is precipitated by acetic acid. This precipitate by acetic acid is insoluble in an excess of the acid, and thus differs from the precipitate of syntonin (see p. 138).

The mucus that comes from the stomach is in most cases translucent and unpigmented. Pigmented mucus very rarely comes from the stomach itself; only in cases of prolonged chronic catarrhal inflammation of the mucous membrane. When pellets of pigmented mucus are present in the vomit, the source of the mucus is the bronchial tubes (as in bronchitis and pulmonary tuberculosis).

The *presence of pus.* Pus is in some cases present in the vomited matters. It is usually the muco-pus from the pharynx or bronchial tubes brought up by vomiting excited by cough: and this is readily diagnosed by the muco-pus occurring in pellets or in strings, many of which are pigmented. Pus may be present in the vomit and come from the rupture of an abscess into the œsophagus or into the stomach. Abscesses of the stomach itself are rare (see Gastritis Mycotica, Chapter IX.); and in the diffuse form of suppuration, pus may be constantly present in the stomach contents. Its diagnosis is made by a microscopical examination; pus-cells being seen in various stages of degeneration.

The *presence of blood.* It must be borne in mind that blood may be present, diffused through the mucus in the vomit, and yet come from the lungs, the cough exciting the vomiting. The appearances are much the same as in slight cases of hæmatemesis (see Chapter XIII.). Blood from the stomach is present in the vomit in varying amounts. When the bleeding is profuse and arterial, it is bright red and clotted; when the bleeding is slower but still profuse, it is brought up in black clots; with still slower hæmorrhage, when the blood remains a long time in the stomach, it is changed in colour, turning black or like "coffee-grounds." With slight hæmorrhage, the blood may be seen either as red specks in the vomit, or red or blackish streaks in the vomit, or diffused through the mucus. The diagnosis of blood in the vomit may in some cases be difficult. (1) The microscope may at once decide the point by the demonstration of red blood corpuscles, and not infrequently these are found when there is no distinct naked-eye evidence of the presence of blood, a fact which is of importance as showing the necessity of a systematic microscopical examination of vomited matters, especially in cases of ulcer. (2) The guaiacum-test may show the presence of blood. Two or three drops of tincture of guaiacum are added to a little of the stomach contents in a test-tube, and ozonic ether poured on to the surface; a blue colour develops where the two liquids meet. As a test of the presence of blood in the stomach contents, it is, however, not very trustworthy, since a similar reaction is given by many vegetable substances, *e.g.* potato, and by bile and saliva. (3) In the case of the vomiting of the appearance of coffee-grounds, no diagnosis of the presence of blood can be made by the microscope, by the guaiacum-test, or by a direct spectroscopic examination. "Coffee-ground" vomiting may have to be diagnosed from vomit containing tea or coffee drunk a short time before, or from vomit containing bile. The last is readily diagnosed by the colour reaction with nitric acid (Gmelin's Test); the first two conditions, as a rule, present no difficulty in diagnosis. In coffee-ground vomiting, the red corpuscles are broken up and the hæmoglobin is transformed into insoluble hæmatin. The diagnosis of blood may be made by the application of two tests: (*a*) the formation of crystals of hæmin; (*β*) the demonstration of the presence of iron. (*a*) *Hæmin crystals* are prepared in the following manner. Take a little of the black sediment and mix it with a little common salt on a microscope slide; add one or two drops of glacial acetic acid, cover with a cover-glass and heat over the spirit lamp, until bubbles begin to form. A microscopial examination shows, if blood is present, the characteristic reddish brown oblong crystals of hydrochlorate of hæmin. This test is said to fail in some cases with the stomach contents even when blood is present. (*β*) The *presence of iron* is demonstrated by the formation of Prussian blue. The patient must of course not be taking any preparation of iron for the test to be of value. It is performed in the following way: to some of the black sediment in a porcelain capsule, a small quantity of potassium chlorate is added, and a few drops of strong hydrochloric acid. The mixture is heated over the flame, and on the addition of a few drops of potassium ferrocyanide solution (5 per cent), Prussian blue is developed, if blood is present.[1] This test is a very delicate one.

Melæna.—Blood in the stools is diagnosed from its appearance. When mixed with the motion, as in bleeding from the stomach and small intestine, it is dark, giving the motion a chocolate colour; and may thus be distinguished from the black stools when iron is taken, or the bluish-black stools following the administration of bismuth. In any case of doubt, a microscopical, spectroscopical, or chemical examination settles the point.

4. *Estimation of the total degree of Acidity of the Stomach Contents,* i.e. *the Acidity due to Acid Salts, to Acids in combination with the Proteids, as well as to the Free Acids.*—It is desirable in all instances to do this, as in certain cases the degree of acidity is proportional to the amount of irritation of the stomach.

In the stomach contents, as vomited or withdrawn by the stomach pump, the free acidity may be due to hydrochloric acid alone, or to hydrochloric acid in addition to one or other of the organic acids, lactic, butyric, acetic; or to one or other of these organic acids alone. In the presence of some proteids, such as those in muscle fibre, in white of egg, etc., some of the free acid combines loosely with the albuminous substance, rendering this acid. These acids in combination with albuminous substances are readily neutralised by alkalies, although they behave under certain circumstances somewhat differently to the uncombined acid.

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[1] See Korczynski and Jaworski, *Deutsche med. Wchnschr.*, Leipzig, 1887, Nos. 47-49.

Before the total acidity is estimated, the presence of free acid must be determined. Blue litmus paper will show whether there is any acidity at all; and if the reaction is well marked, free acid is present. But the colour of litmus is also changed by acid salts, as well as by free acids, and therefore it is not an accurate test for free acidity.

Phenol-phthalein is also used for testing whether a particular substance is acid or not. The alcoholic solution is yellow, and is turned deep red by alkalies, and bleached by acids. Like litmus, however, it does not necessarily indicate the presence of free acid, since acid phosphates and acid compounds of organic bases and of albuminous substances give an acid reaction with it.

For the detection of free acids, whether due to hydrochloric acid or to organic acids, or to both, two tests may be used.

1. *Congo-red* is turned blue by hydrochloric acid, and violet by organic acids. The test is best applied by means of Congo-red paper, which is readily made by dipping strips of white filter paper into a saturated watery solution of the dye, and allowing them to dry. When Congo-red paper is dipped into the vomit or liquids removed from the stomach, it is turned a deep blue if free acid is present. Although the difference in reaction between mineral and organic acids, the first giving a blue, the second a violet colour, is evident when the test is applied in a pure solution of either a mineral acid or an organic acid, yet no diagnosis of the kind of acidity, whether mineral or organic, of the stomach contents by means of Congo-red can be made, owing to the difficulty of distinguishing between the different shades of blue and violet.

If the Congo-red paper is made with a weak solution of the dye, hydrochloric acid turns it to a light (Cambridge) blue. The test is best applied with paper dipped in a saturated solution of the dye.

2. *Benzo-purpurin.*—Test-papers are made by soaking strips of filter paper in a saturated solution of the dye and drying. With hydrochloric acid the purple colour is turned dark blue, and this is not removed by shaking with ether. With organic acids (butyric or lactic) the colour becomes brownish black, and the colour is removed by ether; a mixture of these acids and hydrochloric acid gives a similar colour (von Jaksch).

Having settled the question of the presence of free acids, the total acidity of the stomach contents may be estimated. In doing this, the vomit or stomach contents must not be filtered as an incorrect estimation would be made, but they must be well shaken in a bottle or flask until all the particles are broken up. There is no difficulty if the solid matter of the vomit consists chiefly of undigested food, but if much mucus is present, as it is in some cases of gastric catarrh, it is impossible to do anything with the stringy mucus, and so this is best strained off through well-washed butter muslin. Although the result as regards total acidity is not quite accurate, it is sufficiently near the mark for ordinary purposes.

The method of determination of the total acidity is as follows[1] (see also Toepfer's method, p. 134):—

To 20 cc. of the stomach contents add three or four drops of a saturated alcoholic solution of phenol-phthalein, and dilute with water to 300 cc. Place 150 cc. of this mixture in each of two flasks, and place them side by side on a sheet of white paper. To one of the flasks add decinormal solution of sodium hydrate,[2] until a red colour appears; the exact time of appearance can be determined by comparison with the liquid in the other flask. When a pinkish tinge appears, the acid liquid is neutralised. A control estimation may be made with the second flask.

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[1] See Gamgee's "Physiological Chemistry of the Animal Body," vol. ii. p. 498, 1893. Also Martius and Lüttke, "Die Magen-Säure des Menschen," p. 50.

[2] Decinormal solution of sodium hydrate is of the strength of 4 grammes of pure sodium hydrate to the litre of distilled water. The sodium hydrate must be pure, and made from sodium. This weight of sodium hydrate (4 grammes) will exactly neutralise 3·65 grammes of hydrochloric acid.

The result may be expressed in two ways:—

1. In terms of hydrochloric acid. If 50 cc. of decinormal sodium hydrate are required to neutralise 100 cc. of the stomach contents, this would be equal to 0·18 gramme per cent hydrochloric acid, since 3·65 grammes hydrochloric acid are neutralised by the 4 grammes of soda in a litre of the decinormal solution.

2. It was suggested by Ewald that the acidity might be expressed simply by a number, which would be the same as the quantity of decinormal sodium hydrate solution requisite to neutralise 100 cc. of the gastric contents. Thus, if 50 cc. of the soda solution neutralised 100 cc. of the stomach contents, the acidity of the latter would be expressed by the figure 50. It would certainly simplify matters if this mode of reckoning the acidity of the stomach contents were universally adopted. The figures could readily be converted into terms of hydrochloric acid, since a decinormal solution of sodium hydrate is a liquid of a constant strength; and 100 cc. exactly neutralise 0·365 gramme of hydrochloric acid.

3. *Distinguishing Tests and Reactions for the Free Acids which may be present in the Gastric Contents.*—The reactions with litmus and with phenol-phthalein only demonstrate that a liquid is acid or alkaline; those with Congo-red or benzopurpurin show the presence of free acids.

When free acids are present they may be hydrochloric acid alone, or organic acids—lactic, butyric, acetic—alone, or both hydrochloric, and one or other of the organic acids named.

Butyric and acetic acids differ from hydrochloric and lactic acids in that they are volatile, and may thus be driven off by heat. The free acidity of the stomach contents may thus be divided into two classes:—

1. *The fixed acidity* due to hydrochloric or lactic acid.
2. *The volatile acidity* due to butyric or acetic acid.

1. *Fixed acidity* due to hydrochloric acid or to lactic acid.

(*a*) *Tests for Hydrochloric Acid.*—Many colour reactions have been used for the detection of hydrochloric acid, many of which are untrustworthy when applied in investigating the stomach contents.[1]

Methyl-violet. — A solution of methyl-violet (the dye commonly used for staining bacteria) is turned blue by free hydrochloric and other mineral acids; afterwards the solution becomes green and then loses its colour. Organic acids, unless in large amount, do not affect the colour of methyl-violet. This reaction is a delicate one for hydrochloric acid in pure solution; but it is interfered with by the presence of albumin and peptones, of leucin, of acid phosphates, of saliva, and of mucus. It is quite untrustworthy when applied for testing the presence of free hydrochloric acid in the stomach contents, and was the test used by van der Velden in his work on cancer of the stomach, from which he concluded, and many after him, that hydrochloric acid was always absent from the stomach contents in these cases. Cahn and von Mering have effectually disposed of the value of the methyl-violet reaction as a test for free hydrochloric acid in the stomach.[2] A reference to their work will show that they obtained the reaction when no hydrochloric acid was present (in one case), and in many cases no reaction was obtained, although as much as from 0·05 to 0·15 per cent of hydrochloric acid was present.

Colouring Matter of Bordeaux Wine and of Bilberries.—Uffelmann[3] recommended these two tests for free hydrochloric acid.

A mixture is made of 0·5 cc. of red Bordeaux wine (ordinary claret), 3 cc. of alcohol, and 3 cc. of ether; this is nearly colourless. If a nearly equal bulk of solution of hydrochloric acid (even as little as 0·045 to 0·05 per cent) be added, a rose colour appears even in the presence of albumin, peptone, and salts.

The second test is more delicate, and may be applied in test-papers. Bilberries are crushed with a little water, and the mixture shaken with amylic alcohol, which dissolves out the colouring matter. The alcohol is poured off the watery mass and allowed to soak into strips of filter paper. The paper is stained blue or greyish blue, fainter than blue litmus.

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[1] For a full account of these, see Martius and Lüttke, *op. cit.* p. 38.

[2] *Op cit.*, *Deutsches Arch. f. klin. Med.*, Leipzig, Bd. xxxix. p. 233.

[3] "Die Methoden des Nachweises freier Säuren im Magenhalt," *Ztschr. f. klin. Med.*, Berlin, Bd. viii. pp. 392-406.

With 0·024 per cent hydrochloric acid, the paper is stained a rose-red colour, which is not removed by ether. Organic acids give this reaction, but only in solutions more concentrated; lactic acid, for example, in a solution of 0·4 to 0·45 per cent, acetic acid in 0·5 to 0·6 per cent, and butyric acid in 0 45 per cent solution. Uffelmann states that this proportion of organic acids is never found in the stomach contents; this is, however, not the case (see Cahn and von Mering, *op. cit.*). In any case, the reaction with the colouring matter of bilberries, although delicate, is not so serviceable as others.

The tests just described are not so certain as the three following ones, which are sure indicators of the presence of free hydrochloric acid in the stomach contents.

Tropæolin 00 is a yellow aniline dye, which is slightly soluble in water, and more soluble in a mixture of alcohol and water, giving a yellow solution. When saturated, or nearly so, the solution is reddish yellow. With mineral acids this yellow solution becomes a pinkish red, and gives a brilliant reaction if the acid is in any quantity; with organic acids, the solution becomes slightly red. Although, however, there is this distinction between the colour reaction with mineral and organic acids, when these are in pure solution, no absolute reliance can be placed on it as a means of discovering the presence of hydrochloric acid or of organic acids in the stomach contents, unless the test is applied in a particular way. Like Congo-red, 00 tropœolin may be used as a test for free acidity, whether due to organic acids or to hydrochloric acid or to both; it does not give any reaction with acid salts or with compounds of hydrochloric acid and albuminous substances. This test is applied by shaking up one gramme of the substance with 30 cc. of methylated or rectified spirit and filtering. One or two drops of the clear reddish-yellow solution may be added to a little of the gastric contents in a test-tube; if free acid be present, the yellowish solution becomes red.

In applying tropœolin as a test for free hydrochloric acid a drop of the solution is placed on a white porcelain plate, and allowed to evaporate to dryness at the body temperature; when dry a drop of the liquid to be tested is added: a violet or purple tint develops if free hydrochloric acid is present. As little as 0·005 per cent of hydrochloric acid gives this reaction, and performed in the manner described, the test is a sure indicator of the presence of free hydrochloric acid.

Gunsberg's Phloroglucin-vanillin Test.[1]—The test solution is as follows:—

Phloroglucin	2 grammes
Vanillin	1 gramme
Alcohol	30 cubic centimetres.

If a drop of this yellowish solution be mixed with a drop of a dilute solution of hydrochloric acid and evaporated to dryness in the water bath, a rose-red colour is developed, due to the formation of red crystals. As little as 0·05 per cent of hydrochloric acid will give this reaction, and the development of the reaction is not interfered with by the presence of organic acids, of albumin, or of peptones. Organic acids, even in strong solution, do not give the reaction; so that the test is a very trustworthy indicator of free hydrochloric acid, and is especially applicable to the examination of the stomach contents.

Boas' Resorcin Test.—Boas' solution is made by dissolving 5 grammes of resorcin with 3 grammes of cane-sugar in 100 cubic centimetres of weak spirit. The test is applied in exactly the same way as the previous one (Gunsberg's), and gives a similar rose-red coloration if free hydrochloric acid be present. A difference of delicacy between these tests cannot be detected; neither solution gives a reaction with organic acids, and both detect a small quantity of hydrochloric acid (0·05 per cent). Gunsberg's solution is very expensive; while Boas' has the advantage of being cheap.

According to Salkowski and Kumagawa,[2] the reaction with Gunsberg's solution is not obtained with the acid compound of hydrochloric acid and leucin which exists in some cases in the stomach contents, as Richet pointed out; leucin, however, does not exist in the stomach in such large quantities as to damage the practical value of the test. It must be remembered that all these colour-reactions for hydrochloric acid indicate only the free acid, and that they are not given by hydrochloric acid in combination with proteids. The absence of the colour-reaction when applied to the stomach contents does not prove that no hydrochloric acid is secreted, because some secretion may have occurred and the acid may have combined with the proteids present. In this case, the amount of acid must be estimated by one of the methods described on p. 134.

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[1] Gunsberg, *Centralbl. f. klin. Med.*, Bonn, 1887, No. 40.

[2] *Virchow's Archiv*, bd. cxxii. pp. 235-352.

(*b*) The other fixed acid that may be present in the stomach contents is lactic acid; and this is more constantly present than either of the volatile acids, butyric or acetic.

In solution the presence of lactic acid is detected by two reactions:—

1. Uffelmann's reaction.[1] The test solution used is composed as follows:

Carbolic acid solution (1 in 20)	10 cc.
Water	20 cc.

Add one or two drops of liquor ferri perchloridi.

A clear amethyst-blue liquid results, the colour of which is changed to a clear yellow or a greenish yellow by a trace of lactic acid. As little as 0·01 per cent of the acid will give the reaction; but the test is not very satisfactory if the acid be present in large quantity, and it must be applied to the residue of the ether extract of the stomach contents, since the presence of hydrochloric acid, of proteids and phosphates, interferes somewhat with it. Tartaric and citric acids give a similar reaction; but hydrochloric acid, acetic, and butyric acids simply discharge the colour of the solution.

2. In another reaction, more delicate than Uffelmann's, the test solution is made by adding one or two drops of liquor ferri perchloridi to 50 cc. of water. This mixture is almost colourless, but when lactic acid is added to it it develops a yellow coloration. Hydrochloric, acetic, and butyric acids do not give this reaction.

These tests as applied to the yellowish contents of the stomach, which have a large amount of finely broken up undigested residue, often fail, and therefore they are best applied to the ethereal extract in the following manner. Fifty cubic centimetres of the gastric contents are concentrated to about

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[1] *Op. cit.*

10 cc. in a capsule over a water-bath, and the concentrated liquid is shaken, *after cooling*, with about 50 cc. of ether. The concentration gets rid of any volatile acids present, and the ether dissolves out the lactic acid, leaving the hydrochloric acid in the residue. The ethereal extract is then allowed to evaporate, and any residue is dissolved in a little (5 cc.) water. If this watery solution is acid, the lactic acid tests may be applied; if it is not acid, no appreciable quantity of lactic acid is present in the stomach contents.

When the stomach contents are not yellow, but a nearly colourless liquid, and readily filtered, the tests for lactic acid may be directly applied, or they may be applied to the ethereal extract which is obtained by shaking the liquid with twice its bulk of ether, and pouring off the ether after allowing the mixture to stand. The ether must, as before, be allowed to evaporate, and the extract taken up with water.[1]

2. *Volatile Acidity of the Stomach Contents, due to Acetic or Butyric Acid, or both.*—No colour reactions are available for testing for these acids. Their presence is discovered partly by the smell and partly by their characteristic volatility and reactions.

One method is as follows[2]:—

Place 50 cc. of the filtered stomach contents in a small retort or Erlenmeyer's flask connected with a Liebig's condenser, which leads into a flask to receive the distilled product. Distil the liquid by means of a bunsen-burner, taking care that the contents of the retort do not char; distil until three-fourths of the liquid have passed over. Dilute again to 50 cc., and distil until three-fourths have passed over. Butyric and acetic acid will be collected in solution in the receiving flask, leaving lactic and hydrochloric acids in the retort.

In the solution of the volatile acids, butyric acid is readily recognised, especially when a little of the liquid is evaporated carefully to dryness, by its pungent smell of rancid butter, and by the following reaction: To a small quantity of the liquid, add a small quantity of alcohol, and two drops of strong sulphuric acid; on heating for a short time, there is a characteristic smell of butyric ether, like that of "pine-apple rum." Acetic acid may be recognised by its smell, by the blood-red colour which it gives with solution of perchloride of iron, and by the formation of acetic ether in the same manner as butyric ether. Acetates also give the cacodyl reaction, viz. a nauseous pungent smell, developing when potash and a little arsenious acid are added to the liquid containing them, and the mixture evaporated in a test-tube. The smell of cacodyl is readily recognised and not easily forgotten. Butyric acid is much less soluble in water than acetic acid; when first mixed with water it forms little oily drops, which on shaking disappear.

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[1] Boas has introduced a test for lactic acid, consisting in the recognition of one of its oxidation-products, viz. aldehyde, which may be tested for by Nessler's reagent. It is, however, too elaborate for purely clinical investigation (see Boas, *op. cit.*, part i. p. 178).

[2] Cahn and von Mering, *op. cit.*

By neutralising with barium and calcium carbonate, a butyric or acetate is formed, and the amount of acidity due to these acids estimated. A readier method, however, of estimating the total acidity due to volatile acids is to estimate the acidity of the distillate of the stomach contents, obtained in the manner above described, by means of decinormal solution of sodium hydrate, and reckoning the percentage of acidity as acetic acid.

Thus, as an example: if 70 cc. of distillate is obtained, place two portions of 20 cc. each in two beakers, and add to each three or four drops of solution of phenol-phthalein. If 5 cc. of decinormal solution of soda are used to neutralise, this would equal an acidity of 0·016 gramme acetic acid, or in 70 cc., the total distillate, of 0·056 gramme acetic acid. To obtain this distillate, 50 cc. of the gastric contents were used, therefore the amount of volatile acidity in 50 cc. is equal to 0·056 gramme acetic acid, or 0·112 gramme per cent.[1]

For all practical purposes it is sufficient to reckon the volatile acidity as acetic acid.

A shorter method of estimating the volatile acidity of the stomach contents is first to estimate the total acidity in the manner described (p. 126). Then take 50 cc. of the contents and evaporate to small bulk in the water-bath, add distilled water, about 50 cc., to the concentrated liquid, and again evaporate to small bulk. Finally, grind up the residue with a measured quantity of distilled water and estimate the acidity again. The difference between this acidity and the total acidity gives the volatile acidity of the stomach contents, which may be reckoned as acetic acid in the manner described.

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[1] 40 grammes of sodium hydrate neutralise 32 grammes of acetic acid, *i.e.* the 4 grammes of sodium hydrate contained in the litre of decinormal solution would neutralise 3·2 grammes of acetic acid; or, in other words, 100 cc. of the decinormal solution corresponds to 0·32 gramme of acetic acid.

Determination of the Total Quantity of HCl and of Organic Acids in the Stomach Contents.—The determination of the percentage of each acid present in the stomach contents is a matter of great difficulty, and can only be carried out by complicated methods, and by those skilled in chemical manipulation.

Toepfer[1] has suggested a method of estimating the total acidity, as well as the acidity due to hydrochloric acid and that due to organic acids, which he recommends for use in clinical investigation. The following reagents are required :—

1. Decinormal solution of sodium hydrate.
2. Phenol-phthalein, 1 per cent solution in alcohol.
3. Sodium alizarin sulphonate, 1 per cent solution in water.
4. Dimethyl-amido-azobenzene, 0·5 per cent solution in alcohol.

The method is applied as follows :—

Place from 5-10 cc. of the stomach contents in three beakers, A, B, and C. To beaker A add two drops of the phenol-phthalein solution, and add decinormal sodium hydrate solution until the liquid is quite dark red and the colour is not deepened by the addition of more alkali. To beaker B add three or four drops of the alizarin sulphonate solution, and add decinormal sodium hydrate until the first appearance of a violet tint, equal in depth to that given by four drops of the alizarin solution and 5 cc. of 1 per cent solution of sodium carbonate (Na_2CO_3). To beaker C add three or four drops of the dimethyl-amido-azobenzene solution : and add decinormal soda until the last trace of red has disappeared, leaving only a yellow tint.

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[1] G. Toepfer, *Ztschr. f. physiol. Chem.*, Strassburg, Bd. xix. pp. 104-122. Abst. in *Journ. Chem. Soc.*, London, June 1894 : Abst. II. p. 262.

The result of the titration of C = free hydrochloric acid.
The result of the titration of B = total acidity.
The result of the titration of (B − A) = loosely combined HCl acidity.
The result of the titration of B − C and (B − A) = organic acidity and that due to acid salts.

The determination of the percentage of HCl in the stomach contents may be done by several methods; for the exact performance of which text-books on *Physiological Chemistry* may be consulted.[1] The method of Cahn and von Mering consists in getting rid of the volatile acids (butyric and acetic) by distillation, then removing the lactic acid by ether; and finally, adding fresh cinchonine to the liquid in order to combine with the HCl. The cinchonine hydrochlorate is removed by chloroform, and the amount of chlorine in it estimated. The large quantity of ether which is used makes the process very expensive, and the results are not as accurate as those of other methods. Of the other methods, Sjöqvist's is the most easily performed. The principle on which it rests is that the free HCl of the stomach contents combines with added barium carbonate when evaporated together to dryness; barium chloride is thus formed, which may be separated by means of boiling water. The amount of barium is estimated by forming the sulphate, and thus the amount of chlorine with which it has combined.

Lüttke's method of estimating the amount of hydrochloric acid (both free and in combination with proteids) in the gastric contents is one of the best at present devised. The process of analysis consists, first, in estimating the total quantity of chlorine, and then the quantity of chlories after the free hydrochloric acid has been driven off by heat: subtracting the second result from the first, the amount of chlorine not in combination is obtained, and from this the percentage of hydrochloric acid can readily be calculated.

1. *Decinormal solution of silver nitrate*, containing 16·997 grammes of pure silver nitrate dissolved in 900 cc. of 25 per cent nitric acid; 50 cc. of the liquor ferri persulphatis (B.P.) are added and the mixture diluted with water to 1000 cc.

2. *Decinormal solution of ammonium sulphocyanate*

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[1] *See* Halliburton's "Chemical Physiology and Pathology," and Gamgee's "Physiological Chemistry," vol. ii. p. 499. Also Martius and Lüttke, *op. cit.*

(NH_4CNS), containing 7·6 grammes of the salt to the litre. This solution added to the silver nitrate solution gives in excess a reddish colour. It must be standardised against the silver solution, so that on mixing 10 cc. of each solution a reddish colouration just appears.

The determination of the total chlorine is made as follows: to 10 cc. of the shaken gastric contents add 20 cc. of the silver solution; shake and set aside for a few minutes. Dilute with water to 100 cc., and filter through a dry filter. The precipitate on the filter contains the chlorine in combination with silver, while the filtrate contains the excess of silver solution used. This is estimated by taking 50 cc. of the filtrate and adding the sulphocyanate solution until a red colour appears. The number of cc. used multiplied by two, equals the number of cc. of silver solution used in excess. The amount of silver in combination with the chlorine, and thus the amount of chlorine, are now easily calculated.

The determination of the chlorine in the form of salts is made as follows: place 10 cc. of the gastric contents in a platinum capsule and evaporate to dryness. Ignite the residue until it no longer burns with a flame, and extract the chlorides by grinding the residue with hot water up to 100 cc. Filter, and add to the filtrate 10 cc. of the silver solution. The excess of silver used must be estimated and the amount of chlorine in the same way as in the first process.

The chlorine present in the form of chlorides is subtracted from the total chlorine, and the resulting number multiplied by 0·0365 gives the amount (weight) of hydrochloric acid present in 100 cc. of the gastric contents.[1] The whole estimation takes about an hour to perform.

For estimating the amount of volatile acids present, the only method is to distil the stomach contents in the manner described on p. 132, and determine the acidity of the distillate.

For estimating the lactic acid present, the volatile acids must first be got rid of, and the lactic acid removed by repeated extractions with a large excess of ether and the acidity of the extracts estimated (Cahn and von Mering). A second method is by means of the "*coefficient de partage.*" This latter method depends on the fact discovered by Berthelot,[1] that if a watery solution of an organic acid and a mineral acid be shaken with an equal volume of ether, a certain amount of the organic acid dissolves in it, the amount varying with each organic acid, but remaining constant for the same acid at a given temperature. The proportion of the amount of acid dissolved by the ether to the amount of acid in the watery liquid below the ether is the "*coefficient de partage.*" The method requires great care, as the temperature must be kept constant.

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[1] See Gamgee, *op. cit.*

It must be said that for ordinary clinical purposes, these methods are too complicated, and that with regard to ordinary cases the following points are the only ones that need be examined:—

1. The total acidity of the stomach contents (p. 126).
2. The determination of the presence of free acids (p. 124).
3. The determination of the presence of free HCl by means of the colour tests described (p. 128).
4. The determination of the presence of lactic acid by shaking the concentrated gastric contents with ether, and testing by means of the colour tests for the acid in the ether residue (p. 131).
5. The determination of the presence of the volatile acids (p. 132).

6. *Determination of the Presence of the Products of Digestion in the Stomach Contents.*—It is important in some cases to detect the presence of albumoses and peptones. The presence of these bodies in a liquid which has regurgitated, or has been vomited or removed from the stomach shows that the organ is capable of secreting pepsin and hydrochloric acid. A necessary precaution must be observed in ascertaining whether peptonised food is taken or not; if it is, no deduction can be made that the peptones found were formed in the stomach.

Albumoses and peptones are very rarely absent from the contents of the stomach when these are vomited or removed by the stomach-pump. This is true if the vomit is very liquid or if it has been ejected some time after food. When, however, vomiting occurs soon after food, albumoses and peptones may not be discoverable. In cases of obstruction of the lower end of the œsophagus the regurgitated liquid, which is sometimes in large enough quantity to be examined, does not contain these substances, since the food has never entered the stomach. In such a case the examination of the liquid for albumoses and peptones is of great importance from the point of view of diagnosis.

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[1] Berthelot et Jungfleish, *Ann. de chim.*, Paris, tome xxvi. p. 396, 1872; also Richet, *op. cit.*

Method.—For testing for the proteid products of digestion the liquid must be clear. If the stomach contents have a large amount of undigested residue or mucus, it is best to strain them first through well-washed butter-muslin and then filter through a thin white filter-paper. This filtrate may be either very acid or slightly acid or neutral.

If very acid, it must be neutralised by solution of sodium hydrate; litmus paper being used to determine when it is exactly neutralised. If phenol-phthalein or 00 tropœolin is added to the liquid, the pink colour which develops on neutralisation interferes with the reactions to be performed. The precipitate which falls on neutralisation is syntonin; it is soluble in excess of alkali, and may be again precipitated by acid, in an excess of which it is also soluble. The precipitate must be filtered off before the tests for albumoses are performed.

In this filtrate, or in the filtered stomach contents which are very slightly acid or neutral, albumoses and peptones may be tested for. The presence of these bodies is shown by adding a drop of solution of copper sulphate (1 per cent) to a little of the liquid in a test-tube; this colours it a very faint greenish tinge, which becomes a bright pink colour on the addition of solution of potash, which must be added carefully (biuret reaction). A forty per cent solution of potash or the liquor potassæ of the B.P. may be used. Instead of a 1 per cent solution of copper, Fehling's solution may be used; a mere trace being added to the liquid to be tested by means of a glass rod, and solution of potash being subsequently added. It is sometimes recommended to pour the liquid to be tested on to the surface of some Fehling's solution in a test-tube, the pink colour being developed at the junction of the liquids if albumoses and peptones be present. This is a very rough method, and is not comparable in delicacy to the first one described.

The biuret reaction is common both to albumoses and peptones, and as a rule it is not necessary, clinically, to do more than this test. If, however, it be required to know whether albumoses are present, the following reactions may be performed.

1. To a small portion of the liquid add neutral ammonium sulphate ($(NH_4)_2SO_4$) until no more dissolves on shaking; if albumoses are present they are precipitated and usually float on the surface of the liquid.

2. Add nitric acid to the clear stomach contents drop by drop; if a precipitate forms it is due to proto-albumose. It redissolves on heating and comes down again on cooling.

In testing for ordinary clinical purposes, *i.e.* to determine whether any digestion has occurred, it is sufficient to neutralise the filtered stomach contents for the presence of syntonin, and to perform the biuret test for the presence of the final products of digestion, albumoses and peptones.

7. *Determination of the Presence of Pepsin in the Stomach Contents.*—The presence of pepsin in a liquid is determined by the fact that if hydrochloric acid be added to the amount of about 0·2 per cent, the liquid transforms coagulated egg-albumin into albumoses and peptones. The actual manipulation varies somewhat according to the composition of the stomach contents. These must in all cases be strained and filtered until a clear solution is obtained. This clear filtrate usually contains albumoses and peptones; it may be very acid or only slightly so, or it may be neutral.

1. If only slightly acid or neutral, the method employed is as follows: A solution of egg-albumin is made by separating the white of one egg, cutting it up with scissors and shaking with 200 cc. of water, to which two drops of acetic acid (B.P.) have been added. Some flocculent shreds of membrane are allowed to settle, and the clear supernatant albuminous solution is poured off. Twenty cubic centimetres of this are placed in a large test-tube or flask and coagulated firmly by heat, a small drop of acetic acid being added, if necessary, to ensure complete coagulation. The coagulated albumin is now ready.

To this mixture of coagulated albumin and water add the following in order:—

Acidum hydrochloricum dilutum	20 drops.
Distilled water	70 cc.
Filtrate of stomach contents	10 cc.

Make a similar mixture as a control experiment, but before adding the filtrate of the stomach contents, boil it so as to destroy any pepsin present. Place both test-tubes in the warm chamber at 40° C. for one hour. If the stomach contents contain pepsin, the coagulated albumin in the first test-tube will be completely or nearly completely dissolved; and the biuret reaction obtained will be much more brilliant in colour than in the second test-tube. The object of having a second test-tube containing the boiled filtrate of the stomach contents is that these already contain albumoses and peptones. The reactions of these bodies are not destroyed by boiling, but the pepsin is, so that at the end of the time of digestion the amount of albumoses present in the first test-tube (which is equal to the amount formed plus those already present) may be compared to the amount already present in the gastric contents.

Carried out with the precautions given, this method is a very trustworthy one for determining the presence of pepsin in the stomach contents.

2. If the stomach contents are very acid, they must be strained and filtered as before. The acidity may be due to HCl alone, or in association with the organic acids—lactic, acetic, butyric. Before using such a liquid for testing for pepsin, it must be neutralised and any precipitate filtered off. The subsequent procedure is precisely similar to that just described. The results are not quite so accurate as in the first instance, if organic acids are present; but there is no difficulty even in this case of determining whether active pepsin is present in the gastric contents or not.

If a more accurate method is required, the neutralised filtrate of the stomach contents may be concentrated to small bulk *in vacuo* over sulphuric acid, and then thrown into a large excess of alcohol: a precipitate falls which contains the pepsin. This precipitate is removed and dissolved in water, the solution being tested as regards its digesting action in the same manner as above described.

The *presence of the curdling ferment* is readily determined by neutralising the stomach contents with dilute soda solution, and adding 10 cc. to a similar quantity of milk. The mixture must be neutral. If the mixture is placed in the warm chamber, coagulation of the milk occurs in about fifteen minutes, when the ferment is present.

EXAMPLES OF THE EXAMINATION OF THE STOMACH CONTENTS.

As examples of the examination of the stomach contents, three cases may be taken which illustrate the chief varieties of liquids vomited or removed from the stomach in diseased conditions: a case in which the vomited matters were neutral or nearly so; a second case in which they were hyperacid, due to an excess of hydrochloric acid; a third case in which they were hyperacid, due to an excess of the organic acids of fermentation.

CASE I.—Male, aged forty-four years, with the symptoms of subacute gastritis. The vomit examined was ejected during the stage of convalescence, the patient being on a liquid diet of milk and beef-tea without any solid food whatever.

The vomit was small in quantity, and consisted of a large amount of very tenacious and stringy mucus, and a small quantity of liquid in which there were one or two clots of casein. The mucus was strained off through a Chamberland's porcelain filter and 40 cubic centimeters of a clear yellowish liquid obtained.

This liquid gave a brilliant biuret reaction, showing the presence of a large quantity of albumoses and peptone.

Acidity.—It was very faintly acid to litmus paper. Tropœolin gave no reaction for free acid, and ether extracted no acid body. The liquid was practically neutral.

Digestive Activity.—A digestive mixture was made as follows:—

Filtrate of stomach contents	15 cc.
Water containing two drops of HCl	75 cc.
Coagulated egg-albumin	a small quantity.

This was placed in the incubator at 99° F. for one hour, at the end of which time nearly all the albumin was dissolved, and the remainder dissolved on heating the liquid. The acid liquid gave a dense precipitate on neutralising (= syntonin) and an intense biuret reaction.

The vomit in this case therefore contained a large quantity of active pepsin; no hydrochloric acid; but albumoses, peptone, and a large quantity of mucus. It is illustrative of the condition of the stomach in cases of acute gastritis during recovery; a large amount of mucus being still secreted, but the gastric glands are regaining their secretory activity, although the amount of hydrochloric acid secreted is deficient. The analysis may also illustrate another point, viz. that when the remains of a meal is delayed in the stomach, the hydrochloric acid diminishes (*i.e.* is absorbed), while the pepsin and peptones are still present; hydrochloric acid being more readily absorbed than the former.

CASE II.—Female, aged 48 years, with the symptoms of gastric irritation and of atony of the stomach; associated with chronic bronchitis of a mild type. She was on a diet of bread and milk. The vomit examined was ejected an hour and a half after breakfast.

General Appearance.—Thick and white, separating into two portions; a finely divided solid matter below, and a turbid liquid above. The deposit is starch, since it gives a deep blue colour with iodine. Some fat floats on the surface. There is no sour smell; no mucus.

Peptones.—The liquid gives a brilliant biuret reaction.

Acidity.—Very acid to litmus paper; Congo-red paper is stained a brownish black blue.

Tropœolin 00 gives a red coloration for free acid, and also the purple colour of the evaporation test; Gunsberg's solution gives a red colour. Free hydrochloric acid is therefore present.

The tests for *lactic acid* were negative when applied directly to the vomit. The ethereal extract of the vomit contained fat, but no free acid, and gave neither of the tests for lactic acid. Lactic acid was therefore absent.

Volatile Acidity.—50 cc. of the vomit was twice distilled. The distillate was not acid, and consisted only of water. No volatile acids were present.

Total Acidity.—20 cc. of the vomit were diluted to 300 cc. with water and divided into two portions of 150 cc., each portion was neutralised by 9 cc. of decinormal solution of sodium hydrate (phenol-phthalein being used as an indicator). Therefore 100 cc. of the vomit would be neutralised by 90 cc. of the soda solution; and as 100 cc. of this solution corresponds to 0·365 gramme of HCl, 90 cc. would correspond to 0·329 gramme HCl, which is the percentage of acid in the vomit.

The results of the analysis therefore are:

A large amount of undissolved starch, with peptones and fat and a high percentage of HCl (0·33 gramme per cent), the only acid found. This shows that in this particular case that one and a half hours after a meal, consisting almost solely of starch and milk, the acidity of the stomach had already risen to far above the normal; digestion was proceeding, but the hyperacidity caused vomiting. It was a case of hypersecretion of hydrochloric acid.

Case III.—Female, aged seventy, with enormous dilatation of the stomach; no definite tumour to be felt or signs of carcinoma ventriculi. The patient was on a diet of peptonised milk, with one egg to the pint of milk, five ounces being given every four hours; peptones were also given every four hours. The stomach contents were removed by the pump about one hour after the last feeding with peptonised milk.

General Character of Liquid Removed.—Frothy, yellow, turbid, with a fine yellowish deposit on standing, and a thin pale liquid above.

Peptones.—Well marked biuret reaction.

Sugar.—Fehling's solution readily reduced.

Microscopically.—Large quantities of saccharomyces, with groups of sarcina, a few bacilli, and a few cocci, some in chains.

Bacteriological Examination.—Cultures were made in neutral bouillon containing 3 per cent of sugar. Only yeast was obtained, which in subcultures somewhat rapidly degenerated (see Fig. 16).

Free Acidity.—Congo-red paper turned black.

Tropœolin: both the liquid and evaporation tests were negative.

Gunsberg's test: negative.

Boas' resorcin test: negative.

Free acids were therefore present as shown by Congo-red paper, but there was no hydrochloric acid.

Uffelmann's carbolic iron test and the ferric chloride test gave doubtful reactions for lactic acid, the colour of the stomach contents interfering with the colour reaction.

Total Acidity.—Estimated in the manner described: 78 cc. of $\frac{1}{10}$ NaHO neutralised 100 cc. of the stomach contents = 0·285 gramme per cent reckoned as HCl.

Volatile Acidity.—50 cc., distilled twice, gave 60 cc. of acid distillate, which was neutralised by 2·1 cc. of $\frac{1}{10}$ normal NaHO, *i.e.* 100 cc. of stomach contents contained a volatile acidity which would be neutralised by 4·2 cc. $\frac{1}{10}$ normal NaHO. The distillate gave no reaction with ferric chloride; acetic acid was absent, but butyric ether was evolved on heating with sulphuric acid and alcohol. Since butyric acid was the only volatile acid present, the volatile acidity was equal to 0·37 gramme per cent butyric acid.

Fixed Acidity.—The ether extract of the residue after the volatile acids had been got rid of contained fat and a free acid, which gave both the colour tests for lactic acid.

The degree of fixed acidity estimated as in the manner described was found to be that 60 cc. $\frac{1}{10}$ normal NaHO neutralised 100 cc. = 0·54 gramme lactic acid.

The fixed acidity estimated after simply evaporating the stomach contents to get rid of the volatile acids (see p. 133), was proved to be of the same degree.

Digestive Activity.—The digestive activity of the gastric contents was tested by the following experiment.

Forty cc. of the stomach contents were neutralised with soda, diluted to 100 cc. and three drops of HCl added. Two hundred cc. of solution of egg-albumin were made from the white of an egg. Fifty cc. were placed in each of two flasks (A, B) and coagulated by heat. To each of these flasks 50 cc. of the neutralised and diluted stomach contents were added, and the mixture in flask B was boiled. Flask B therefore acted as a control in two ways: it was precisely the same mixture as in flask A, containing the same amount of peptones which were found in the gastric contents, but the activity of any pepsin present was destroyed by boiling: therefore the digestion of the albumin in flask A could be compared with that in flask B.

The two flasks were kept at 98° F. for twenty-nine hours. In five hours no difference could be discovered between them. At the end of twenty-nine hours, the albumin in flask A had diminished as compared to that in flask B, but the difference was not great, and the biuret reaction was not more intense in A than in B. The digestive activity of the liquid tested was therefore practically nil.

Summary of the Analysis.—Hydrochloric acid was absent, and the hyperacidity was due to lactic acid (0·54 per cent) and butyric acid (0·37 per cent), derived from bacterial fermentation of the stomach contents. Pepsin was practically absent. The results are illustrative of the characters of the stomach contents in great dilatation of the stomach with bacterial fermentation of the food. It may be added that in this case, as peptones were given with the food in the form of peptonised milk or of solid peptones, their presence in the stomach contents was no sign that any digestion had taken place.

II.—METHODS OF EXAMINATION OF THE DIGESTIVE ACTIVITY, OF THE MECHANICAL POWER, AND OF ABSORPTION OF THE STOMACH.

It has already been pointed out that the examination of vomited matters in many instances gives a correct indication of the digestive activity of the gastric juice in individual cases, since from it may be learnt the facts not only that hydrochloric acid and pepsin are secreted, but that albumoses and peptones, the products of proteid digestion, are formed; *i.e.* that the pepsin-hydrochloric acid of the gastric juice is active. In certain instances, also, the time of vomiting, *i.e.* the period elapsing after the last meal, shows that the mechanical power of the stomach is deficient, and that there is thus delay of food in the organ. In many cases of disordered digestion, however, vomiting is absent or is only an occasional occurrence. In these cases other methods may be employed to determine the activity of the gastric juice, the mechanical power of the stomach, and the rate of absorption.

It must be clearly borne in mind what is required to be learnt by such methods. It is not simply that the results obtained are of physiological interest and importance, but that in each class of cases of disordered digestion it is requisite to know to what degree, if any, the gastric juice (pepsin and hydrochloric acid) is altered in quality or quantity, to what degree the mechanical movements of the organ are affected, and whether bacterial fermentation is present or not, so that the treatment adopted may be on the lines of remedying any deficiency of function discovered. By such methods an attempt has been made to render the treatment of disordered digestion more rational and less empirical. In what respects they fail and in what they succeed will be seen after the methods are discussed.

Methods of Examination of the Gastric Juice.—It has already been pointed out that food is not the only stimulus which excites the secretion of the gastric juice; mechanical, thermal, electrical, and chemical stimuli have a similar action. The introduction of a sound into the stomach excites the secretion, also the drinking of hot or ice-cold water, and the passage of an electric current through the organ by means of a sound introduced into it.

As a chemical stimulus to secretion, a dilute solution of bicarbonate of sodium or of soda acts with the least irritation.

Mechanical and electrical stimuli are not of practical use in obtaining the gastric juice, but both chemical and thermal stimuli may be employed.

Leube[1] recommended the washing out of the fasting stomach with 400 cubic centimeters of lukewarm water, the last portions removed being tested by litmus paper for neutrality, and then injecting 50 cc. of a solution of soda (3 per cent); this was allowed to remain in the organ twelve minutes, and was then removed by washing out the organ with 400 cc. of water. If after twelve minutes the soda solution is neutralised, the HCl secretion of the stomach is normal; but if the secretion is deficient, the solution remains alkaline. The presence of pepsin in the liquid removed can be determined in the manner previously described (p. 139).

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[1] *Op. cit.*

Another method also used by Leube is to inject 100 cc. of ice-cold water through the sound, after the stomach has been thoroughly washed out. After remaining in the organ ten minutes, the liquid and secretion may be removed by washing out with 300 cc. of water. The presence of HCl and of pepsin may be readily determined in the liquid. In some cases, no doubt, as Leube found, these methods are sufficient in themselves to indicate a deficiency in the secretion of the gastric juice; but a little consideration shows that such methods are not the most serviceable that can be employed. They will clearly show whether hydrochloric acid and pepsin are secreted or not; but this is not the chief point required to be known. In most cases of disordered digestion it is not so much a question whether hydrochloric acid and pepsin are secreted, for in only a few diseases are they actually absent; nor is it solely a question whether stimuli, such as an alkali or ice-cold water, will produce secretion, but whether during the whole time that a mixed meal remains in the stomach there is a sufficient secretion of gastric juice to digest the proteids and to reduce it to the condition of chyme. The secretion of the gastric juice must be a continuous one, just as the movement must be continuous for efficient digestion to take place. So that these methods of obtaining the gastric juice are not of much practical importance. Indeed, for testing the digestive activity of the gastric juice secreted, some kind of proteid food must be given, and the effect of the gastric juice on this during a certain period determined.

Test for the Activity of the Gastric Juice.—The most convenient way of doing this test is to give boiled white of egg when the stomach is empty, and to remove the stomach contents in half or three quarters of an hour.

The stomach must be empty, or if not empty washed out before the experiment. The white of one or two eggs is separated from the yolk, thrown into 4 ounces of water, and the mixture raised to the boiling point while kept constantly stirred. In this way the albumin is coagulated in fine flocculi. After cooling it is given to the patient, who is to keep quiet. In from $\frac{1}{2}$ to $\frac{3}{4}$ hour, by means of the stomach sound, 4 or 5 ounces of water are passed into the stomach, and the whole of the stomach contents removed by the aspirator.

An examination of the liquid removed will determine whether the solution of the albumin is complete, *i.e.* whether there is any undigested residue, and whether albumoses or peptones are present by means of the biuret reaction. The degree of acidity and its nature can also be readily determined and the presence of pepsin verified. In estimating the degree of acidity of the liquid it must be remembered that the ascertained figure will be only one half the acidity of the stomach contents, since an equal quantity of water was used to wash out the stomach after digestion. There is no method so accurate as this for determining the activity of the gastric juice in disease. A food stuff is given which the stomach readily digests, and which does not give rise to any organic acidity in the organ. Moreover, although the rate of digestion varies greatly in individuals, yet with such a single meal as that given, in all individuals with healthy digestion, there ought to be an appreciable digestion of the albumin in one hour; while in those with a deficient secretion of gastric juice, digestion will hardly have commenced, the liquid removed being only very slightly acid, containing no albumoses or peptones, and with scarcely any digestive activity.

Such a method as this determines only the activity of the gastric juice; it does not indicate the mechanical power or the absorptive activity of the stomach. In cases of disease it is requisite to know not only the activity of the gastric juice, but to have some indication of the power of the disordered stomach to manipulate the mixed food of a moderately large meal. This process of manipulation includes not only the action of the gastric juice on the proteids of the food, but the effect of the movements of the stomach. With regard to the movements, it cannot be too frequently stated that not only must the circular and churning movements be continuous during the whole period of stay of the food in the organ, but when digestion is completed and the food rendered semi-liquid, the stomach must be capable of propelling the last portions of chyme into the duodenum, so that the organ is emptied. A diminution in the circular and churning movements delays digestion, and the inability of the organ to expel all the food into the duodenum leaves the organ constantly with some liquid in it. It is concerning these two points that information is required in cases of disordered digestion; they are as important as the secretion of the gastric juice.

All these points may be determined by a method which has been of the greatest service in the diagnosis of disordered digestion, viz. the method of a test-meal.

Method of Testing the Activity of the Gastric Juice and the Movements by means of a Test-meal.—The object of a test-meal is to determine:

1. The activity of the gastric juice.
2. The power of the stomach to manipulate the food.
3. The power of the stomach to empty itself in a certain time.

Some of the test-meals which have been introduced do not adequately determine these points. A test-meal ought to be considered as a physiological meal of moderate bulk, composed of proteids, carbohydrates, fats, and water, the constituents of which are readily digested under normal conditions; and inasmuch as the chief function of the stomach as a digestive organ is the action of the gastric juice upon proteid food, it is advisable that the test-meal contain an excess of proteid food stuff, such as meat or white of egg. Ewald's test-breakfast consists of 35 grammes ($1\frac{1}{3}$ ounce) of bread and $\frac{1}{3}$ of a litre of water. This contains an excess of carbohydrates, and does not constitute an accurate test of the power of the stomach to manipulate the solid food or to empty itself after the digestion of a moderately sized meal. But it is of value in determining the amount of secretion of gastric juice in response to the stimulus of food. It may also be used where a large meal could not be borne by a patient; although in such cases it is best to use milk alone or white of egg as the test-meal. A more trustworthy test-meal for ordinary cases of disordered digestion is that used by Leube and others. It consists of a little soup, a large beef-steak (about 5 ounces) and a small bread roll, no water being drunk with the meal, which is to be taken at midday. The patient is allowed to rest after the meal, and the stomach must previously be fasting. After five to seven hours have elapsed the stomach contents are removed by the sound and investigated. Under normal conditions the stomach is completely empty in seven hours after such a meal, containing only a little neutral liquid, with a few flakes of mucus. In five hours there is usually some undigested muscle fibres and starch grains still in the stomach contents, which are acid and contain peptones. In disordered conditions delay of digestion is determined by the fact that in seven hours after such a meal the stomach still contains undigested food, is still acid, and contains peptones. The liquid removed from the organ may be examined by the methods previously described.

The test-meal of Leube's was used by him in the observations previously quoted (Chapters III. and IV.); by its use a fairly complete knowledge of the functions of the stomach in any particular case of disease may be obtained. It is not always necessary to allow the food to remain in the stomach seven hours, in five hours a little of it may be removed and examined.

Methods for Testing the Mechanical Power of the Stomach.—The changes in the mechanical power of the stomach are of great importance in disease, and a simple method of testing it is greatly to be desired. Ewald has used the following method. Salol, which is a compound of salicylic acid and carbolic acid, is decomposed into its component parts by the pancreatic juice as soon as it passes from the stomach into the duodenum. The salicylic acid is excreted in the urine as salicyluric acid, and is recognised by a solution of ferric chloride, which gives a reddish violet colour with it. Ewald gives 1 gramme (15 grains) of salol in a gelatin capsule just after the midday meal. In healthy individuals the salicylic acid secretion appears in the urine in sixty to seventy-five minutes. Several observers have, however, obtained very varying results, the time of the appearance of the secretion in healthy individuals varying between thirty and ninety minutes. The conclusion is therefore that the test is of service only when the time of excretion is much delayed. Attention has already been drawn to one fallacy in such a method, viz. reckoning the absorptive activity of one organ in terms not only of the absorption but also of the excretion of a chemical substance by another organ supposed to be healthy. This was referred to in discussing the iodide of potassium test of absorption by the stomach (Chapter III. p. 107). The salol test is much more complicated, inasmuch as the substance has to be expelled from the stomach, to be decomposed by the pancreatic juice, to be absorbed by the intestinal mucous membrane and to be excreted by the kidney; and for the test to be of use it is necessary to suppose that the pancreas, intestinal mucous membrane, and kidneys are performing their functions in a normal manner.

Other considerations show that the salol test is quite valueless as a gauge of the mechanical power of the stomach. The gelatin of the capsule is soon dissolved in the gastric juice, the gramme of salol is set free and is practically insoluble in the acid liquid; the fine particles are passed through the pylorus with some of the liquid and decomposed in the duodenum. The quantity of liquid that passes through the pylorus during digestion of food in the stomach varies, as has been shown, very greatly, according to the consistency of the digesting food, and therefore the rapidity with which the salol would be sent into the duodenum would vary. But what is required to be known about the mechanical power of the stomach is not that a small amount of a substance can pass through the pylorus in a certain time, but that in a certain limited period the stomach can empty itself of the mixed food of a moderately sized meal. The salol test is no reaction for this, and is indeed from a clinical and practical point of view valueless; for although if the reaction time were greatly prolonged (say to 120 minutes) it would indicate diminution of the mechanical power of the stomach, yet in mild cases of disorder in which it is most important to determine the degree of mechanical power of the stomach, the test would not show the condition.

Klemperer[1] has used a test of the mechanical power in which 100 grammes of oil are placed in the stomach by means of the sound. In two hours the remainder of oil is removed by adding some water to the stomach and aspirating. The amount of oil present in the washings is dissolved by ether, the ether evaporated and the oil weighed. Seventy to eighty per cent of the oil ought to be discharged into the duodenum by a healthy stomach in two hours.

There is no doubt that this is a fair test of the mechanical power of the stomach: it has the disadvantage, however, of being disagreeable, and of entailing rather long chemical manipulation, and is a method more adapted for research than for application for ordinary clinical purposes.

Methods for Testing the Absorption of the Stomach (see p. 105).—It may be said in conclusion that in determining the alterations in the functions of the stomach in disease, much may be learnt from an examination of vomited matters, not only as regards the chemical processes of digestion, but as regards bacterial fermentation of the stomach contents. The quantity of vomit and the period elapsing after a meal may indicate delay of food in the organ, and diminution of the mechanical powers of the stomach.

In the great majority of cases of disordered digestion, it is not necessary to apply the tests for the functions of the organ except in those prolonged cases which appear but little amenable to treatment of any kind, and in cases of suspected cancer. In such cases it is of great value both from the point of view of diagnosis and of treatment, that an accurate knowledge of the processes occurring in the stomach during digestion should be obtained. It is therefore not only legitimate, but it is beneficial to the patient that the digestive activity of the gastric juice should be determined to see whether the constituents of the gastric juice (HCl and pepsin) are secreted; or that the digestive activity as well as the mechanical power should be determined by giving a somewhat larger meal, such as Leube's test-meal.

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[1] *Deutsche med. Wchnschr.*, Leipzig, 1888, No. 47.

Instruments to be used in Investigating the Stomach Contents.—The stomach contents are removed by means of a sound which is passed into the organ, and which has attached at its outer end either the stomach-pump or an aspirator.

The *stomach sound* is best made of indiarubber, not too soft or too hard; it must be from 80 to 90 cm. (32 to 36 inches) long, and from 1½ to 2 cm. in diameter. Its end must be slightly conical, perforated, and with two side openings.

To withdraw the stomach contents, the ordinary pump may be used: it is, however, a powerful instrument, and is best replaced by an aspirator. The ordinary "medical" aspirator, such as is used for tapping the pleura, serves very well, the outer end of the stomach tube being attached to one of the tubes of the bottle. The procedure is exactly the same as in paracentesis of the chest, the bottle being first exhausted by the air-pump. Boas has devised a useful modification of the ordinary indiarubber ball syringe, in which, by the insertion of a valve between the stomach sound and the syringe, the stomach contents are readily withdrawn and emptied through the syringe into a vessel (Fig. 17). Expression of the stomach contents may be resorted to by pressing upon the abdomen, if the patient can bear it. It must not be done, however, if the patient is suffering from any severe illness.

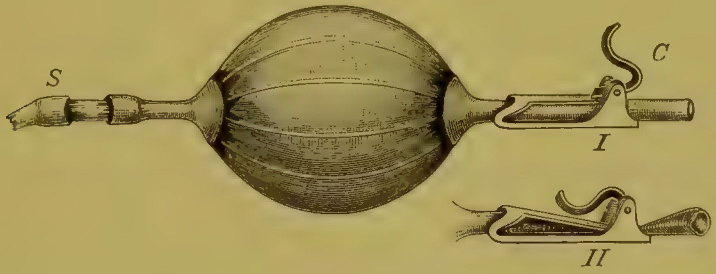

FIG. 17.—Boas' Stomach Aspirator. *S*, connection with stomach sound by a piece of glass-tubing; *C*, a spring clip; at *I*, open; at *II*, closed.

After using the stomach sound, it must be first well washed in cold water by allowing a stream to pass through it, then washed in warm water, and in 1 in 20 carbolic acid solution, finally in warm water again. It must be well washed before being again used. The method of passing it is very simple. Moistening the end with some glycerine, the patient is placed in the sitting posture with the head thrown slightly forward;

the end of the sound is now passed to the back of the pharynx, along the posterior wall of which it is allowed to glide. Telling the patient to take a deep breath, and then to swallow, the sound may be gently pushed onwards, and once in the œsophagus it is easily swallowed. Accidental entrance of the sound into the larynx shows itself by coughing and dyspnœa; it must be withdrawn immediately and passed farther backwards.

Conditions in which the Stomach Sound is not to be Passed.—Before the stomach sound is passed an examination of the patient must be made to see if any serious disease of the thorax is present. It is contra-indicated to pass the stomach sound:

1. When there are any signs or symptoms of thoracic aneurism present.

2. When there is a severe wasting disease or a disease tending to syncope: advanced pulmonary tuberculosis, carcinoma, or serious cardiac disease.

3. When there has been recent bleeding from any part.

4. In cases of great debility or in patients of advanced age.

5. As regards diseases of the stomach: in all cases of ulcer the introduction of the sound into the organ is dangerous, and to be avoided if possible. In advanced cases of carcinoma it is useless as a means of diagnosis, and in many cases of functional disorder it is unnecessary to use it, although not in itself dangerous.

CHAPTER VI.

THE SYMPTOMS REFERABLE TO INDIGESTION OF FOOD WITH OR WITHOUT ANATOMICAL CHANGES IN THE WALL OF THE STOMACH.

In discussing the pathology of indigestion of food, it was seen that the causes which lead to the disturbance of normal digestion in the stomach were various.

1. There are alterations in the secretion of the gastric juice, either in a diminution of the total quantity secreted, or in a diminution of the hydrochloric acid; or, on the other hand, in an increase of the amount of hydrochloric acid secreted. Such changes would affect the chemical processes of gastric digestion, viz. the digestion of the proteids by the pepsin-hydrochloric acid, and the consequent setting free of many articles of food (fat, etc.) which are not digested in the stomach.

2. There are, moreover, alterations in the movements of the stomach, which are sometimes increased, but more commonly diminished. The increased movements are directed to overcome pyloric obstruction, or occur in certain cases where there is excessive irritability whereby the food is rapidly passed through the stomach and intestines. As a rule the movements are diminished, and this diminution leads to delay of food in the organ and its consequences.

3. Alterations in the absorptive power of the stomach cannot be accurately determined. Diminution of the absorptive power would affect chiefly the sugars which are either taken with the food or are formed from starch by the saliva, and the non-absorption of sugar would provide a food for bacterial fermentation.

4. Besides alterations in the secretion of the gastric juice, and in the movements and absorption of the stomach, there are changes in the blood supply and in the processes of innervation which are of great importance in disease. As regards the blood supply there may be anæmia or congestion; and as regards the innervation, there may be either irritability or diminished excitability (irresponsiveness of the stomach). With irritability of the stomach, various reflex effects are observed—*gastric reflexes*—the clinical importance of which is very great, although a complete explanation of their causation is not forthcoming.

5. Lastly, food acts not only as a direct cause of the alterations described, but even when not directly the cause the changes are aggravated by the food taken. Moreover, the presence of food, and especially of carbohydrates, is the fuel for bacterial fermentation.

To obtain any clear idea of the processes of digestion in disease, the symptoms which are referable to these several pathological conditions must be considered as far as is possible, in order to be able in any individual case to recognise the symptoms which may be ascribed directly to indigestion of food in the stomach; to separate reflex effects on the normal gastric functions from reflex effects arising from disorder of the gastric functions; and to differentiate the effects of disordered gastric functions from those arising in the other parts of the alimentary tract. Thus certain symptoms referable to the stomach may be either part of a disorder of the organ or may be a reflex or direct effect of some disease in a distant part, as, for example, when vomiting is the result of uterine, renal, or cerebral disease, when eructation of gas (flatulence) is intestinal in origin, or when nervous symptoms arise not referable to disordered digestion.

In a case, moreover, in which the symptoms are referable directly to the stomach, the effects produced by disorder of function of the organ have to be carefully separated from those which are the results of local or organic disease—ulcer, carcinoma or inflammation—for although in the majority of instances these organic diseases produce definite symptoms as well as disordered digestion, yet in some cases the diagnosis is extremely difficult owing to the absence of definite symptoms.

In discussing the symptoms referable directly to disordered function or to organic disease of the stomach, a little consideration makes it clear that they are divisible into two classes: in one of which the symptoms are direct, arising from changes in the organ; in the other of which they are indirect, *i.e.* they are reflex effects arising from the stomach and resulting in symptoms referable chiefly to the nervous system. To these classes may be added a subsidiary one, in which the symptoms are referable to the effects of disordered digestion in the stomach, but also to an effect on either the salivary secretion or the intestinal functions.

I.—SYMPTOMS REFERABLE DIRECTLY TO THE STOMACH.

1. *Sensations in the Stomach Region.*

(*a*) *A Sensation of Emptiness* which before food is often associated with pain (gastralgia), or which occurs after food in rapid digestion and in rapid emptying of the stomach contents. The first sensation is relieved by food, the second only partially so.

(*β*) *Pain in the Epigastrium* is of two kinds, according as it occurs when the stomach is empty or when it contains a meal.

In an *empty stomach* one form of pain has just been mentioned (gastralgia) which may occur in severe paroxysms. Other forms occur, viz. severe pain in acute gastritis, whether due to inflammation, to poisons, or to the invasion of bacteria (infective gastritis).

Pain after Food, or Pain aggravated by Food, is the usual form of epigastric pain arising from the stomach. It occurs in two forms, localised and diffuse. The diffuse form is associated with hyperacidity and bacterial fermentation, as in cases of gastric irritation and dilatation of the organ. It is also observed in inflammation of the stomach, as in catarrh and the other forms of gastritis. Localised pain is observed in ulcer, sometimes in carcinoma of the organ: it may be associated with dorsal pain and tenderness. Pain in the epigastrium due to a distended transverse colon is not infrequently mistaken for pain due to a stomach condition, as well as the pain and superficial tenderness present in abdominal neuralgia and muscular rheumatism.

(γ) Pain in the left hypochondrium and the lower part of the left axilla is in many instances due to a dilated and full stomach, as well as to a distended colon or enlarged spleen.

(δ) A sensation of fulness, weight, or discomfort in the stomach region without actual pain, occurring directly after meals or some hours after. This is associated with gastric irritation or hyperacidity, with a simple deficiency of movement of the organ, with dilatation of the stomach and bacterial fermentation and distention of the organ with gas. The direct cause, as a rule, of these sensations is the partaking of a meal which is disproportionate to the digestive capacity of the disordered stomach. The bulk of the meal may be too large for even a normal stomach, or it may contain a large amount of indigestible matter (cellulose, fat, starch), or too large a quantity of organic acids or salts, condiments, alcoholic drinks, or other food accessories. In many instances, as will be seen, the discomfort arises one to three hours after the largest meal, indicating that the symptoms are referable to the increasing acidity and to defective movements; for at that time the normal acidity due to HCl and the abnormal organic acidity (due to food or fermentation) is rapidly rising, and the movements of the disordered stomach are diminishing.

2. *Eructation and Vomiting of Stomach Contents.*

(*a*) *Acid Eructations* (*Pyrosis*).—These occur after food, within half an hour or from two to four hours after the meal. They are sometimes more distressing towards the end of the day, after three meals have been taken, than after breakfast. There are varieties of acid eructations, most of which are dependent on gastric irritation. Acid eructations do not occur in dilatation of the stomach, if this is well marked, for the simple reason that the dilated stomach cannot expel small quantities of liquid upwards; vomiting occurs, but not eructation of liquid. The acid fluid eructated may be the normal acid stomach contents, as in cases where a very large meal has been taken, or where there is irritability of the stomach; during the process of digestion the food acting as an irritant to the stomach, and causing eructation. The fluid may be hyperacid due to an excess of hydrochloric acid, as in cases of gastric irritation; or to hydrochloric acid and an excess of organic acids. As a rule when there is well marked bacterial fermentation of the food, there are no acid eructations, since the stomach is usually dilated; but there is no doubt that fermentation of food does occur in some cases, merely as an occasional accident, where there is chronic indigestion of food. In this case the acids eructated would consist partly of hydrochloric, but chiefly of lactic and butyric acids.

The most common varieties of acid eructations are those which occur in healthy individuals from over-feeding and those occurring in gastric irritation, where there is an excess of hydrochloric acid secreted. As a rule acid eructations occur in a full stomach; even if they occur a long time after a meal, the stomach still contains a large quantity of food owing to its delay in the organ.

(β) *Eructations of Neutral and Alkaline Fluids.*—By some, the term pyrosis, or water-brash, is applied to this condition, but it is clear that, if retained at all, pyrosis, from its derivation, ought to be applied to acid eructations, which are frequently very " fiery " in character.

Neutral and alkaline fluids, brought up from the stomach, have various modes of origin. In the first place the neutral or slightly alkaline liquid may simply be the saliva swallowed after the food has been expelled into the duodenum; this occurs in some cases of gastric irritation and hyperacidity, the stomach condition acting reflexly on the salivary glands (see p. 116). Secondly, the eructated fluid may be the exudation into the organ in cases of mechanical congestion due to portal obstruction or to dilatation of the right side of the heart. This is not a common condition, but it does occur in some cases, especially of mitral valvular disease. Thirdly, a common cause of the eructation of neutral or alkaline liquid is the delay of food in the stomach, which reacts in the following manner. After a meal in such individuals the processes of digestion progress normally up to a certain period, the food being broken up and the proteids being digested by the pepsin-hydrochloric acid; and when the chemical processes are completed, most of the chyme is expelled into the duodenum, but not all, since the motor power of the stomach is deficient. What remains behind becomes completely digested, and remaining in the stomach some time, the acid is absorbed, leaving a nearly neutral fluid containing peptones. It is this fluid which is eructated, and which by its long stay in the stomach acts as an irritant. Such cases are observed in gastric catarrh and in gastric insufficiency. A last, and not infrequent source of alkaline fluid in the stomach is the regurgitation of bile and pancreatic juice into the organ through a flaccid pylorus. This occurs chiefly in motor deficiency (atony) of the stomach.

(γ) *Eructations of Gas: Flatulence* (see p. 97).—During normal digestion in the stomach and small intestines, but little gas is generated from the digesting food. In the stomach some carbonic acid is liberated by the gastric juice from the carbonates taken with the food; and in the small intestine, the acid chyme decomposes the carbonates of the pancreatic juice, liberating some carbonic acid gas. The amount found, however, is small, and gives rise to no symptoms. In disease, as has been fully discussed, a large quantity of gas, chiefly consisting of hydrogen and carbonic acid, may be generated in the stomach or small intestine by the bacterial fermentation of carbohydrates. Conditions of digestion in the stomach influence bacterial fermentative processes in the small intestine (p. 116). Thus, if the stomach contents are but slightly acid when expelled into the duodenum, bacterial fermentation is more likely to take place in the small intestine than if the stomach contents are normally or abnormally acid. All the conditions which lead to bacterial fermentation in the small intestine are not known, but the fermentative processes that do take place produce lactic acid, butyric acid, and carbonic acid and hydrogen gases. The practical point is that even when no bacterial fermentation occurs in the stomach, it may occur in the small intestine, and indeed that the gas eructated from the stomach may come from the intestines, regurgitating through the pylorus. This is undoubtedly the case when the eructated gas contains sulphuretted hydrogen and marsh gas. This and other varieties of flatulence may be tabulated in the following manner:—

1. Flatulence due to accumulation of small quantities of gas, chiefly carbonic acid, which are generated from time to time in the stomach and small intestine, and not being absorbed, not being propelled onwards owing to the motor deficiency of the organs, are eructated by the stomach, often towards the end of the day. This is a very common form of flatulence, especially in the middle-aged and the old.

2. Flatulence due to swallowed air, which enters the stomach partly with the food and partly with the swallowed saliva. Saliva in the stomach is also a cause of flatulence, since the carbonates it contains are decomposed by the acid of the gastric juice setting free carbonic acid.

3. Flatulence due to the regurgitation of pancreatic juice into the stomach, the carbonates it contains becoming decomposed and setting free carbonic acid. This is a very frequent form of flatulence, and occurs in flaccidity of the stomach walls and patency of the pylorus; it usually comes on towards the end of digestion.

4. Flatulence due to bacterial fermentation in the stomach; severe and continuous in cases of dilated stomach, passing and accidental in cases of chronic indigestion of food.

5. Flatulence due to the exchange of gas (CO_2 and N) between the blood and the contents of the stomach and intestines. This takes place normally as "intestinal" respiration, and is supposed to be the explanation of the great evolution of gas occurring after severe pain, such as occurs in attacks of migraine, of biliary or renal colic, or in neurotic individuals. Nothing definite is known as to the source of the gases eructated in such cases, but it is surmised that they are discharged from the blood.

(δ) *Vomiting of Stomach Contents.*—Vomiting of the stomach contents may or may not be a symptom of disorder or disease of that organ. The vomiting centre, which is situated in the medulla, by its efferent tracts controls many different muscles and secretory organs, and by its afferent tracts is connected with many organs and mucous membranes (see Chapter I. p. 17). The causes of vomiting may be various, and may be summarised as follows:—

Vomiting not associated with Disorder or Disease of the Stomach.

1. Due to mechanical causes, as where violent cough, coming on after a meal (in pulmonary tuberculosis, chronic bronchitis, chronic tonsillitis, and in hooping cough) ends by causing the ejection of the stomach contents.

2. Reflex causes of vomiting from parts of the body other than the stomach:—

- (α) *Infective diseases,* especially at the onset in children, are associated with vomiting, which is in relation to the food, occurring after a meal.
- (β) With *severe pain* (biliary and renal colic, dysmenorrhœa) vomiting is observed.
- (γ) *Nauseous smells,* or disgusting sights lead to vomiting, and with some people great emotion has a similar effect. In some, particular sounds are said to cause vomiting.
- (δ) *Cerebral disease* (meningitis and cerebral tumour) is a frequent cause of vomiting; the effect being apparently a direct one on the medulla.
- (ϵ) *Functional disturbance of the nervous system,* such as occurs in hysteria and neurasthenia, is frequently associated with vomiting, which in some cases is co-related with suppression of urine; urea and ammonium chloride being found in the vomit.
- (ζ) *Uræmic or renal vomiting* is not due to a reflex effect of the structural changes in the kidney, but to the formation and retention of poisonous substances, associated with an increase of arterial tension.
- (η) *Vomiting in intestinal obstruction and in peritonitis.*

In the diseases which have been enumerated, the stomach may be the seat of disorder or disease as a more or less accidental occurrence, and its condition may predispose to the vomiting. The diagnosis as to whether vomiting is due to one of these conditions or to disorder or disease of the stomach is not always easy to determine, and will be more evident after the consideration of vomiting in disorder of stomach. With regard to those cases due to mechanical causes, such as cough, the diagnosis is simple and evident; the violent cough supervening on a meal is a sufficient cause for the symptom. So in infective diseases, where there is fever associated with the specific symptoms of the disease, the diagnosis is evident; the vomiting as a rule being only an initial symptom. In cases, however, of cerebral disease, and more especially in hysterical vomiting and in that associated with uræmia, the distinction between the vomiting due to these conditions and to stomach disease may be very difficult, if not impossible. When there is optic neuritis and severe headache (vertical or occipital), the diagnosis is rendered clear; as also in cases of uræmia when there is albuminuria, with a pulse of high tension, an enlarged left ventricle, and retinal hæmorrhages.

Vomiting associated with disease or disorder of the stomach has in many instances peculiarities which allow of its diagnosis. These peculiarities are determined in part by the relation of the vomiting act to the taking of food, and in part also by the chemical and physical characters of the vomit.

Vomiting may be directly caused by food, as in the case of eating an abnormally large mixed meal, or a meal containing a large quantity of indigestible or irritating substances. This kind of vomiting is readily recognised, since it occurs once and has a definite cause, and the ejection of the contents of the stomach relieves the symptoms. In some cases the vomiting occurs almost directly after the meal, but in others it is delayed for five or six or eight hours, the food becoming partly digested but remaining in great part in the organ, owing to a diminution of the movements. Vomiting is also due to hyperacidity of the stomach contents, to the continued retention of food as in dilatation of the organ, to nervous irritability of the stomach, to the presence of inflammation, of an ulcer, or of a new growth.

The acute disorders of the stomach which are associated with vomiting, are acute gastric catarrh, toxic gastritis, and infective gastritis, in all of which the vomiting is repeated, and often continuous, and is accompanied by severe and persistent pain.

The chronic disorders and diseases of the stomach associated with vomiting are neurosis of the organ, gastric irritation, gastric catarrh, ulcer, carcinoma, and dilatation. In these conditions, there is pain caused by food (gastric irritation, catarrh, and ulcer), and independent of food (carcinoma); while in simple dilatation, which is only a symptom of disease, there is no pain if the condition is advanced.

1. In gastric irritation and in gastric catarrh the vomiting occurs after food, from half an hour to two hours as a rule, but the vomited matters vary greatly in the two conditions. Thus in gastric irritation, which is an irritative condition of the stomach due to dietetic irregularities, there is an increased secretion of hydrochloric acid. The vomit therefore consists both of digested and of undigested food, and is hyperacid, due to the presence of hydrochloric acid (Chapter V.). In chronic gastric catarrh, on the other hand, there is a deficiency in the amount of hydrochloric acid secreted, and an increased secretion of mucus; the vomited matters therefore consist of undigested food in a slightly acid mixture, with ropy mucus. If the food has remained some time in the stomach, only a little undigested food is present, but peptones are found, and although free hydrochloric acid may be practically absent, active pepsin is present (see Case I. p. 141).

In ulcer the vomit has, except in the late stages, the same characters as in gastric irritation, but in this case the vomiting is produced in part by the hyperacid or normal stomach contents irritating the open ulcer, and is more immediately connected with the ingestion of food.

2. In carcinoma the vomiting is sometimes due to pain, sometimes to catarrh of the mucous membrane, and sometimes to the fact that the gastric juice is unable to digest the food at all, which acts thus as an inert foreign body, and is ejected. In other cases the vomiting is due to dilatation of the organ (Chapter XVI.).

In dilatation of the stomach, the vomiting and the vomited matters are often very characteristic. In moderate dilatation, from whatever cause, the vomiting may be directly referable to a meal taken, but is usually not so, and the vomited matters may show the characteristics of that in catarrh (viz. an excess of mucus, and a deficiency of hydrochloric acid), or there may be a deficiency of hydrochloric acid simply. In well-marked dilatation of the stomach, the vomiting has no direct reference to a meal, it occurs once a day towards the evening, or every two days or so, when a large quantity of liquid has collected in the organ. The characters of the vomit have already been described, and are shown, in the presence of undigested food, of a small amount of hydrochloric acid and pepsin, and of a large but varying amount of organic acids (see Case III. Chapter V. p. 143).

3. Vomiting in neuroses of the stomach is connected with the ingestion of food, and is characterised chiefly by its irregularity.

(ε) *Hæmatemesis* (Chapters V. and XIII.).—The stomach conditions which produce hæmatemesis are: mechanical congestion caused by portal obstruction, or dilated right side of the heart (*e.g.* in cirrhosis and acute congestion of the liver, in portal thrombosis, in cardiac disease); acute congestion and inflammation, as in the different forms of acute gastritis; ulcer and carcinoma of the stomach. General disease also produces hæmatemesis, such as acute fevers (severe malaria, bilious typhoid, yellow fever, typhus) anæmic diseases (hæmophilia, leukæmia, Hodgkin's disease, purpura, scurvy). Aneurisms may rupture into the stomach or blood may be swallowed. The blood which comes from the stomach may be bright red in colour if the bleeding is sudden and profuse and immediately vomited (as in cases of an ulcer opening a large branch of an artery), or it may be dark in colour, or of the appearance of coffee grounds, due to the action on it of the acid gastric juice. A large amount of blood may be retained in the stomach and undergo these changes, as in the blood which exudes slowly from erosions.

This short summary indicates the symptoms directly referable to the changes in the stomach in disease; but they are only a part of the symptoms which result from disordered digestion. The changes in the process of gastric digestion which have been described as occurring in disease, as well as the pathological changes in the stomach walls, produce symptoms not only referable to the organ, but others which may be called, in general, gastric reflexes. These reflex symptoms in some cases mask the true stomach symptoms, and are those frequently causing the most distress to the patient in chronic cases of disordered digestion.

In many of the symptoms about to be discussed, it is impossible to decide exactly what pathological factor is active in producing them. These symptoms are such as referred pains in the head, chest, or elsewhere; palpitation, cough, and dyspnœa, and a large class of symptoms referable to the nervous system, and shown not only in affections of the higher mental faculties, but of the emotional faculties, and in objective sensations, such as vertigo, buzzing in the ear, etc., and in affections of motion and sensation.

In the explanation of such symptoms, three causes may be considered: (1) either they are caused by the disordered digestion acting reflexly through the nervous system; (2) or they are produced by certain (unknown) toxic products formed during disordered digestion; (3) or they are produced by a disordered metabolism of the tissues caused by the indigestion of food. Reserving the fuller discussion of their cause until the symptoms themselves are considered, it is well to bear in mind that disordered digestion in the stomach frequently occurs in association with other conditions. Thus it may be present in "nervous" individuals, and show certain characteristic features, or it may be present in certain general debilitating and wasting diseases (chlorosis, pulmonary tuberculosis, etc.) which will stamp the disordered digestion with certain features, or it may be associated with disorder of digestion in the small or large intestine, and with disorder of function of the liver and pancreas. To a great extent the symptoms of the disordered digestion can be dissociated from those due to a general disease, but in the case of disordered digestion in the stomach and in the intestine, the question is a much more difficult one, owing to the limited knowledge possessed of functional disorders of digestion in the small intestine, and of functional disorders of the liver and pancreas.

II.—REFLEX SYMPTOMS OF DISORDERED DIGESTION.

1. *Appetite: its Diminution (Anorexia) or Increase (Boulimia).*—Physiologically, appetite or the desire for food is an expression of the need of body for the taking in of food. The physiological explanation of appetite is, however, by no means simple. Thus, normally, appetite is developed when the stomach is empty at a certain time after a meal, and the maintenance of a healthy appetite depends not only on the emptiness of the stomach, but on the regularity with which meals are taken. It is supposed by some that the development of appetite is caused by the secretion of saliva (as in "watering of the mouth"), which, being swallowed, stimulates the secretion of a small quantity of gastric juice. But although this may be the primary stimulus in the development of appetite, yet it must be considered that appetite is a nervous phenomenon, dependent not only on a stomach condition, but on a condition of the central nervous system.

A normal appetite is affected reflexly in many ways. Thus, as is well known, nauseous smells and tastes, as well as disagreeable sights, will take away the appetite, which may also be lost as the result of great pain and mental shock, such as a great grief or a great joy. On the other hand, some smells and sights are appetising, and the increased appetite developed at social functions is so well known as only to be mentioned. Some of these effects on the appetite result from a reflex action through the peripheral nerves of smell, sight, or taste, or of an ordinary sensory nerve, while others result directly from the central nervous system. The physiology of appetite is therefore complicated, and in disease it is important to bear in mind that the appetite may be affected, as in health, in two chief ways—

1. By the condition of the stomach itself.
2. By the condition of the central nervous system, which may be affected either directly by disease or disorder or by some general disorder of the body.

(*a*) *Appetite as Affected by the Condition of the Stomach itself.*

—In not a few cases of disordered digestion, the appetite remains unimpaired or even increased during a certain stage. In these cases, although food causes disturbance and definite symptoms, yet the desire for food recurs at each meal; as patients often express it, "their appetite is good, but they are afraid to eat." Such cases are as a rule those in which disordered digestion is the result of dietetic irregularities. Up to a certain point, the appetite may be cultivated, so that increasing quantities of food and food accessories are taken, and with relish. A continuance of such a habit leads, as has been seen, to disordered digestion, while the desire for food still remains. These cases will afterwards be considered from their clinical aspect under the heading of gastric irritation.

As a rule in chronic disorders of digestion the appetite is impaired. Thus whenever there is continued delay of food in the stomach, so that the organ is not empty when the next meal is taken, there is in time impairment of the appetite. This, however, does not occur in the early stages. In these the appetite for the midday or evening meals is good, while that for breakfast is diminished. The explanation of this common symptom (want of appetite for breakfast) is simple; it is due to delay of food in the stomach during the night. A meal is taken late, in a condition in which the emptying of the stomach is delayed; the patient goes to bed too soon after the meal, and during sleep digestion is still further delayed, so that, often after a restless night, the stomach is not quite empty even in the morning, and indeed vomiting of the stomach contents may occur on rising. Loss of appetite accompanies dilatation of the stomach, whether simple or due to pyloric stricture, or associated with bacterial fermentation of the food.

The appetite is also impaired when there are organic changes in the stomach walls. Thus in acute inflammation (acute gastric catarrh, and other forms of gastritis) the appetite is lost, often completely; in chronic catarrh there is great impairment of the appetite as well as in mechanical congestion of the stomach. In cases of atrophy, whether primary or secondary to inflammatory processes, there is a similar loss of appetite, and anorexia or loss of appetite is an important symptom in cancer. Ulcer of the stomach forms an exception, inasmuch as in many cases, even the majority, the appetite is preserved.

(β) *Appetite Affected by a Condition of the Central Nervous System produced by a General Condition of the Body.*—The effect on appetite of the condition of the central nervous system is a most important point for consideration in disordered digestion. It is frequently stated that the state of the appetite is no criterion of the functional activity of the stomach. This statement is, however, true only within certain narrow limits. In some cases of functional disorder (gastric irritation) there are symptoms of indigestion of food, although the appetite is good or even increased. In ulcer too the appetite may be unaffected. When, however, there is diminished appetite, there is diminished functional activity of the stomach. The conditions which affect appetite through the nervous system are various, and may be considered under the following headings:—

1. A direct effect on the central nervous system due to occupation or disease.

2. An indirect effect due to a general condition of the body.

Healthy occupation, which means a combination of mental and physical exercise, maintains and stimulates appetite; absence of occupation tends to diminish it. Up to a certain point, the increased exercise of the mental faculties increases the appetite; so too with physical exercise. In both conditions there is an increased need of food by the organism. But if the increase of mental or physical exercise is continuous as well as excessive; if it is associated with mental worry or with irregularity in the taking of meals, then the appetite diminishes. In both excessive mental and excessive physical exercise the effect on appetite comes through the central nervous system. This is less apparently so in the case of physical exercise; but in great fatigue or continued and excessive manual labour the chief effect is not on the muscles but on the parts that govern the muscles, viz. the spinal cord and brain. The rest to the central nervous system given by sleep, even of short duration, restores the appetite. Excessive mental exercise is not the only condition which diminishes the appetite. Probably the majority of cases which comes under this heading are due to mental worry and to mental hurry. Those who suffer from periods of worry or anxiety due to their occupations or social surroundings lose their appetite; those who in the race for success are always in a state of mental hurry suffer in a similar way. These effects are intensified in certain individuals, especially women, with an excitable nervous system.

The general conditions of the body which affect the appetite are those producing anæmia and those associated with a chronic wasting disease. In anæmic conditions, in which there is a diminution in the amount of the hæmoglobin and in the number of the red corpuscles, the metabolic processes of the body are not only diminished, but there is an increased excitability of the central nervous system. In such conditions, whether due to chlorosis or to the profound anæmic diseases (leukæmia, lymphadenoma, scurvy, hæmophilia, purpura), or to hæmorrhage, there is anorexia or loss of appetite, due partly to the effect on the central nervous system and partly to the effect on the digestive processes, the secretion of gastric juice and the motor power of the stomach.

In chronic wasting diseases, besides the anæmia produced, there is the increased destruction of the tissues, chiefly fat and muscle, which is shown in emaciation. In malignant disease, in chronic pulmonary tuberculosis, in chronic Bright's disease, and in diabetes, there is increased destruction of tissue. Up to a certain point, increased destruction leads to an increased intake. This is seen in diabetes, where the onset of the disease is often marked by an increase of appetite; but eventually in all the diseases mentioned, the result is loss of appetite: the increased destruction of tissue diminishes the vitality of the tissues and organs and thus affects the appetite.

In both functional and organic disease of the nervous system, the appetite may be affected. There may be an increase of appetite (boulimia, polyphagia), and this is observed in cases of hysteria, of neurasthenia and of mania. There is sometimes loss of appetite; but the two chief effects on the appetite observed in disease and disorder of the central nervous system are a varying appetite (parorexia), at one time great, at another small, and a craving for unsuitable and even harmful substances which may or may not be articles of diet. The latter condition is observed in pregnancy and in mania; the former is a feature of some cases of nervous dyspepsia and of neuroses of the stomach.

2. *Thirst.*—Thirst is one of the symptoms of disordered digestion. It is a desire for water; and, although as a physiological process it is not completely understood, yet one or two facts in connection with it are of importance. In the first place, thirst, or an increased desire for water is developed in those conditions in which there is an increased excretion of water from the body by the skin, kidneys, or intestines, both in physiological and pathological conditions. Again, thirst is not allayed if the water drunk is allowed to flow out of the duodenum by a fistulous opening as soon as swallowed; this has been shown by experiment. The absorption of the water is thus necessary to allay thirst. When thirst is present, its more distressing symptoms are partially allayed by increasing the flow of saliva, which moistens the dried mouth; such as the drinking of a small quantity of an acid liquid which causes an increased flow of saliva.

There is thus a certain proportion between the amount of water and solids in the body which must be maintained; if the amount of water is diminished then thirst is developed. In the diminution of the amount of water in the body, there is a decreased diffusion of salts, and of the excretion products of the tissues (the chemical products of their metabolism). Thus, while the circulation of the blood may not be interfered with to any great extent, there is a diminished interchange between the tissue salts and the salts in the blood, and with the diminished excretion of the metabolic products there is a diminished activity of the tissues. Thus the desire for water, although locally excited by the dryness of the mouth and the emptiness of the stomach, is yet dependent on the condition of the tissues, and no doubt arises in the central nervous system.

The main conditions with which thirst are associated are :—

1. An *increased excretion of sweat*, whether in physiological conditions, such as after great exertion; or in pathological, as in the sweating stage of fevers, or in the sweats of chronic wasting diseases (*e.g.* tuberculosis of the lungs).

2. An *increased excretion of urine*, chiefly in diseased conditions, *e.g.* diabetes mellitus, diabetes insipidus, and other cases of polyuria.

3. An *increased secretion of water by the intestines.* Thirst is present in such conditions when the increased secretion of liquid is rapid, as in acute diarrhœa due to irritants (chemical or bacterial); or when the contents of the stomach and intestines are rapidly passed out of the body, as in lienteric diarrhœa. In some instances, however, of disordered digestion, thirst is present even when a large amount of liquid is not passed out of the body. In these cases it is probable that the liquid is retained in the intestinal tract for some time, and thus is not available for the needs of the tissues. In such cases, there is probably a diminished peristalsis (atony of the intestinal wall) with diminished absorption; in other cases at first, the retention and then the vomiting of the contents of a dilated stomach leads to thirst.

4. Irritation of the stomach by certain kinds of unsuitable food leads to thirst. These are chiefly salted or cured (smoked) foods, together with spices, alcoholic drinks, tea, and other food accessories. A large proportion of salt with the food leads to thirst to a slight extent by acting as an irritant to the mucous membrane, but chiefly by causing an increased flow of liquid from the blood vessels of the mucous membrane into the gut. During normal digestion, the proportion of water to solids in the digesting food in the stomach and small intestine does not vary greatly, this being due to the constant passage of water from the digesting food to the vessels of the intestines, and *vice versâ*. When, however, the saline constituents of the food are greatly increased in proportion to those in the blood and tissues, there is an increased flow of liquid into the intestine, which may remain there for some time, and lead to a temporary condition of thirst. Over-indulgence in spices and alcoholic drinks possibly act partly in this way, but also by acting as

irritants to the mucous membrane, delaying digestion, and by acting as diuretics. That irritation of the gastric mucous membrane does lead to thirst, probably chiefly by delaying the absorption of water, is seen in gastritis, acute and chronic, in which thirst is often a prominent symptom. It may partly be due to the loss of liquid by vomiting (and in incessant vomiting thirst is an observed symptom), but it is not solely due to this, since in cases of chronic gastritis, where there is no incessant vomiting, thirst is not uncommonly present.

It is now necessary to consider other symptoms associated with indigestion of food; symptoms, the exact origin of which it is in many cases difficult, if not impossible, to determine. These symptoms are those referable chiefly to the nervous system, and consist in pain in various parts, and in disturbances of the mental and emotional faculties as well as of the motor and sensory systems. Besides, there are symptoms referable to the circulatory and the respiratory systems, such as palpitation, cough, and dyspnœa.

Pain.—Pain not directly referred to the stomach region is a very common symptom in those subject to disordered digestion. It is of various origin. In some cases it is directly to be attributed to the process of digestion going on in the stomach; in others it is more directly due to the general condition of the patient, either as regards the condition of the blood (as in anæmia) or of the nervous system (as in neurasthenic individuals).

Pain directly dependent on the indigestion of food is referred to the chest, the front, back, or one or other side. The pain in front of the chest and between the shoulders is that most commonly observed. It is a dull pain, not sharp and lancinating like that of neuralgia, and is commonly described as a sense of weight and oppression in the chest. In front of the chest, the pain is not usually associated with local tenderness, but between the shoulders there is not uncommonly distinct and diffuse tenderness over a greater or lesser area. With regard to these kinds of pain, it may be said that they occur chiefly in cases of chronic indigestion of food, and that at first they occur after a meal, in half an hour, or in one or two hours, and diminish or disappear as the stomach becomes emptied. In prolonged or aggravated cases of indigestion these pains, however, are almost constant; and those suffering from them will often deny that they have any relation to meals. Close inquiry will usually, however, elicit the fact that at first they appeared after meals. There are two chief factors in the production of these pains; one consists in the existence of irritation of the gastric mucous membrane by food, either through its inherent indigestibility, or its slow digestion and delay in the stomach. The other factor is that indigestion of food, which is continued over months and years, affects the general condition of the body, and especially the nervous system, whereby the pain is exalted. That the pains under consideration are directly dependent on indigestion of food is shown finally by the fact that they are relieved by treatment directed to the cure of the indigestion.

Pain in the left side, *i.e.* in the lower part of the left axilla, is not uncommonly associated with a dilated stomach, and with flatulence. It may also be due to anæmia or to a loaded colon. Pain in the right side is not only associated with derangement of the liver, but also with a loaded colon. Both these varieties of pain are commonly associated with areas of local tenderness. Distinct neuralgias may occur in the course of indigestion of food, partly referable to the stomach condition. The occurrence of neuralgia cannot, however, be directly attributed to the indigestion; it almost invariably occurs in two classes of patients (omitting the neuralgic pains due to carious teeth), viz. in neurotic (neurasthenic) subjects, or in the anæmic, especially in chlorotic young women. The neuralgia may be over the area supplied by the fifth nerve, or it may be intercostal, in which case it is not infrequently associated with herpes zoster.

Other Symptoms Referable to the Nervous System.—Besides pain, there are other symptoms referable to the nervous system, which not infrequently are the most prominent signs in cases of indigestion of food, and give rise to a great deal of bodily and mental distress. It is in many cases difficult to gauge these symptoms, inasmuch as they may originate primarily in a disorder of the central nervous system, or they may be secondary to indigestion of food, which acts as a peripheral irritant starting them, or they may be associated with local disease or disorder elsewhere than in the stomach.

These symptoms may be classified as referable to (1) the cardiac and respiratory systems, such as palpitation of the heart, cough, and dyspnœa; (2) to the motor system, such as a tired feeling and a sensation of the legs giving way during walking, and also the occurrence of cramps of the limbs and of the abdominal muscles; (3) to sensation, such as tingling and numbness of the extremities; (4) to the special senses, such as muscæ volitantes, a sense of impaired vision, and buzzing in the ear; and (5) to the central nervous system, such as drowsiness, sleeplessness and bad dreams, vertigo, inability to do mental work, and the dread of a fatal seizure, cardiac or apoplectic.

The origin of these symptoms is complex, and in the present state of knowledge, no pretension can be made to an accurate understanding of them. They occur chiefly in individuals usually described as neurotic, neurasthenic, etc., *i.e.* in those in which there is an irritability of the central nervous system. But they do not occur solely in such individuals. They are, for example, most apt to occur at certain periods of life, or in certain conditions of body. They are noticed especially at about middle age, and in women at the menopause; and they frequently occur during pregnancy and lactation, in prolonged anæmia, and in those leading a sedentary life as well as an intellectual one.

Cough and dyspnœa as the result of indigestion of food is more directly referable to the morbid processes going on in the stomach than are the other symptoms. Cough is in some instances due to the eructation of the acid and acrid stomach contents irritating the throat, especially when the tonsils are enlarged or there is chronic faucio-pharyngitis or granular pharyngitis. In some individuals, cough is excited by a dilated or an over-distended stomach, the causation in such a case being partly a mechanical interference with the proper working of the diaphragm. Paroxysms of cough occurring after meals are frequent in those suffering from diseases of the lungs; both in chronic bronchitis and emphysema, and in pulmonary tuberculosis. In the case of chronic bronchitis and emphysema the production of cough after or even during a meal is due chiefly to the fact that in rigidity of the chest the breathing is chiefly diaphragmatic; food in the stomach interferes with the proper working of the diaphragm, hence there is an accumulation of mucus and a deficient intake of oxygen, and cough results, so that the attack of cough is usually brought on by large meals and obviated by small ones. This is not a complete explanation of the violent cough produced in such cases. When cough follows a meal in pulmonary tuberculosis, the incidence of the symptom is not proportional to the extent of disease in the lungs, but is associated with cases in which the cough is habitually violent and in which there is anæmia and nervous irritability. In such cases the violent cough ensuing after a meal must be considered a reflex effect, the peripheral irritant being the food in the stomach, and the afferent nerve the vagus conveying the impulse to the medulla. No other explanation appears probable, and this holds good not only for pulmonary tuberculosis, but also for chronic bronchitis. Violent cough not related to the intake of food is usually present in those cases in which there is anæmia or irritability of the nervous system. It must be borne in mind also, that continued and violent cough renders the respiratory centre in the medulla more irritable, so that a slight peripheral irritation of the throat or other part which would not in the normal state excite cough, brings on a paroxysm, and also that excitation of parts which do not normally lead to cough produces a paroxysm; the stomach is one of these parts.

Dyspnœa is not a common result of the indigestion of food, and when it occurs after a meal, the stomach condition is not the sole or even the chief cause of its production. The ingestion of food may in this case, as in the production of cough, be the peripheral irritation starting the dyspnœa. In cases of simple indigestion of food, dyspnœa is an exceedingly rare symptom, and in such cases, patients rarely make it a subject of complaint. But in three distinct conditions of the body, either the normal digestion of food or, more commonly, its indigestion, may lead to dyspnœa; viz. in anæmia, in asthma, and in cardiac disease. In anæmia and in cardiac disease, the dyspnœa supervening after a meal may be considered chiefly a circulatory effect. In anæmia it is produced by a disturbance of the circulation, due partly to the attraction to the working stomach of too large a quantity of blood which is deficient in oxygen and hæmoglobin, and partly by embarrassing the heart. In cardiac disease, the dyspnœa is probably a more or less direct effect of the distended stomach on the heart, which becomes embarrassed in its action; but a reflex effect on the heart's action through the nervous system may also occur. In asthma a special variety of the disease has been described under the term "peptic" asthma, the attacks of dyspnœa chiefly supervening one to two hours after a meal. There are no clear pathological reasons for making such a division of the disease, for the chief pathological changes in the two varieties are essentially the same. The attacks coming on in relation to a meal must be ascribed to a reflex effect from the stomach through the vagus nerve on the respiratory centre, and the exciting causes belong to the same category as irritating particles drawn into the bronchial tubes. In such cases there is usually indigestion of food, and when this is corrected the dyspnœic attacks diminish in severity.

Palpitation of the heart is a symptom which may be ranged with the foregoing. It is a common symptom in indigestion of food occurring in certain individuals. It occurs most commonly in the indigestion of middle age, especially in women at the menopause, and in the neurotic; but it also occurs in the anæmic, in cardiac disease (chiefly of the mitral valve) and in those addicted to over-indulgence in tea and tobacco. It is ascribable chiefly to a reflex effect through the vagus nerve on the medulla. It is often extremely distressing, and with the sense of weight and oppression over the pericardium or below the left mammary region often leads to the belief of the patient that there is heart disease, which, however, is usually absent or is only present as a coincidence, and not infrequently also it gives rise to a dread of impending apoplexy. Such sensations are commonly associated with chronic indigestion of food, in which there is much flatulence. Another sensation described by dyspeptics is that in which the heart appears to "stand still" or to "turn over" and then begin beating regularly again. This symptom, it may be said, is sometimes associated with an intermittent beat of the heart, an intermittency which is commonly only temporary; but the conditions with which it is almost constantly accompanied are those present in the early or middle stages of gastric irritation. These are the passage of acid or gaseous accumulations through the cardiac orifice or the presence of hyperacid stomach contents. It is perhaps not too much to assume that the sensations ascribed to the heart are more or less due to those conditions, and indeed that in most cases the sensations are really at the cardiac orifice, and in others are reflex effects on the cardiac rhythm.

In organic disease of the heart (valvular disease, fatty degeneration, angina pectoris) indigestion of food sometimes produces serious symptoms. The starting of an anginal attack may be the indigestion of a particular meal, and in fatty heart fatal syncope may follow a large meal.

The effects of indigestion of food on the nervous system affect not only motility and sensation, but also the higher cerebral functions, intellectual and emotional. It is difficult to determine how far these symptoms are purely nervous or how far they may be due to vascular disturbance; in some, both elements are evidently combined.

Drowsiness is a very common symptom complained of by dyspeptics, usually, but by no means always, in those of middle age. At first the drowsiness occurs after a meal, but in the later stages of the indigestion of food, it has no direct relation to a meal. The drowsiness after a meal is allied to the condition of repose which is necessary for the good digestion of large meals by healthy people. This repose means mental inactivity, and is perhaps to some degree dependent on the increased blood supply to the abdominal viscera (not only to the stomach) during the digestion of a large meal. In the dyspeptic, the mental and bodily repose leads to a drowsiness which is only momentary, but is pathological. In the middle-aged and old a large meal actually induces sleep, lasting from ten to thirty minutes. All these cases may possibly be explained by a deficient supply of blood to the brain, a deficiency which, in indigestion of food, is possibly increased by the co-existing disturbed and increased vascular supply to the organs of digestion. In the later stage of indigestion of food, the drowsiness has in many instances no direct relation to a meal, but in these cases the stomach is probably never empty between meals, there being well-marked delay of food in the organ.

Sleeplessness is associated in individual cases with drowsiness; patients complain that although they are drowsy during the day they cannot sleep at night. In other cases there may be no drowsiness, and yet there is sleeplessness. Inability to sleep is due to many causes—to organic disease of the various organs, the heart, central nervous system, etc., to severe pain from whatever cause, to occupation involving intellectual strain, worry or excitement, to over-exercise in the healthy, and especially in the nervous and the anæmic. Although these conditions have to be investigated in each individual case to discover the cause of the sleeplessness, yet in disordered digestion the insomnia is in the majority of instances to be ascribed to the presence of food in the stomach during sleep. Thus it is noticed in those who partake of a large meal one or two hours before going to bed. This may occur as an occasional incident in those quite healthy, but it is more commonly observed in those suffering from disordered digestion to a greater or less degree. It is also present in disordered digestion when it cannot be ascribed to a large meal taken shortly before going to bed. In these cases the stomach also contains food; there is delay of food which is slowly digested, or the digestion of which has progressed to a certain degree and has then been arrested. During sleep, normal digestion is delayed or arrested altogether; and the sleeplessness may be directly ascribed in these cases to the presence of undigesting food, which is often indigestible and usually acid. It therefore acts as an irritant, affecting the central nervous system. It not only produces sleeplessness, but restlessness, snatches of disturbed sleep, and horrible dreams. Similar results occur with a full bladder, and in the later months in some cases of pregnancy. As soon as, during the night, the food has left the stomach, being either vomited in the acute cases, or passed into the duodenum in the milder cases, the patient falls to sleep, which is often profound, waking in the morning with a metallic or bitter taste in the mouth. Some patients do not get rid of all the food from the stomach during the night, and after a disturbed rest they wake and vomit the remainder of the contents of the stomach, which are often small in quantity and excite violent retching. This condition exists not only in the chronic alcoholic, but in those in whom the disordered digestion cannot be ascribed to over-indulgence in alcoholic drinks.

Vertigo is a symptom which may occur after a single food debauch, but, as a rule, there is a prolonged history of dyspeptic symptoms preceding the attacks of vertigo. Its origin must be ascribed not merely to the actual process of indigestion of food in the stomach, but also to the effect of the chronic indigestion of food on the nutrition of the body, whereby the nervous and vascular systems are affected. In cases where vertigo is constantly associated with emptiness of the stomach, the symptom is evidently the result of a reflex nervous action, since like the gastralgia of similar origin it is relieved by the taking of food. The exact nervous mechanism is, however, not easily explained. In a second class of cases, vertigo does not appear to be related to any particular state of the stomach, except that, as a rule, it comes on after food is taken, on rising from the table or not for some time afterwards. In both classes of cases there are symptoms of chronic indigestion of food, which is the prime cause of the vertigo, since this is relieved when the indigestion is cured or benefited. There are other facts which tend to show that there is no great distinction to be drawn between these two classes of stomach vertigo.

The vertigo of a disordered stomach—*vertigo a stomacho laeso* [1]—usually occurs long after food has been taken, when the stomach is "empty." It is sudden in onset, and although the patient may fall to the ground there is no loss of consciousness. There may be nausea and occasional vomiting during the attacks, as well as sensations other than vertigo in the head, such as a feeling of emptiness in the head, a sensation of an iron ring round the head, or even in some cases one of impending apoplexy. There are always symptoms referable to indigestion of food in the stomach; pain after food in the chest or in the epigastrium, flatulence or acid eructations, and the occasional vomiting of food which may be mixed with glairy mucus. The bowels are usually constipated, but there may be diarrhœa alternating with constipation. In such cases, however, it is incorrect to speak of the stomach as empty. Living in the ordinary way, taking three meals a day, such patients never have an empty stomach, there is a delay of food in the organ, due partly to inefficiency of the chemical processes of digestion, partly to weakness of the muscular wall, so that when the patient takes a second meal the stomach is not resting, but contains some liquid which is occasionally vomited. So that the term vertigo of an empty stomach is a misnomer. Food relieves in mild cases, as the entrance of food stimulates the organ to increased work for a time; but food gives no permanent relief unless the indigestion be treated. The severe form of stomach vertigo which has just been discussed is not so common as the milder forms, which are indeed very frequent in women and in men of about middle age who are the subjects of indigestion of food. Such attacks of vertigo are nearly always initiated by some exertion, such as rising from the table, or walking soon after a heavy meal, especially in those who lead a sedentary life involving brain-work. They may also be initiated by reading, and by following moving objects with the eye. The vertigo is sudden in onset, but in these mild cases the patient does not usually fall down. There is no loss of consciousness, and the vertigo which is in no definite direction, is of but momentary duration. Although at the time very alarming to the sufferer, they are not serious, and, like the more severe attacks, are relieved by treatment directed to the relief of the indigestion.

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[1] *Vertige stomacal: vertigo per consensu ventriculi.* See Trousseau, "Lectures on Clin. Med.," *New Syd. Socy.*, 1870, vol. iii. p. 537.

Vertigo is a symptom of conditions other than those due to the stomach. Some of these are purely nervous in origin, while others are due to an effect of the circulation on the nervous centres.

Of nervous origin may be cited the vertigo of Menière's disease, of epilepsy (*petit mal*), and of tumours of the brain. In Menière's disease the symptoms very frequently are similar to those of the severe cases of stomach vertigo; thus in both there is giddiness, with buzzing in the ears, and nausea or vomiting. But whereas in Menière's disease there is in the attacks a sensation of turning round in a determinate manner, this is not so evident in cases of stomach vertigo. Menière's disease is also in many instances associated with deafness in one ear, and is a condition which, although capable of being relieved, is not cured; whereas, when deafness is associated with stomach vertigo, the connection is only accidental, and stomach vertigo is eminently curable.

In epilepsy (*petit mal*) the attacks of giddiness are associated with loss of consciousness (it may be of only momentary duration) or with the performance of determinate acts of which the patient is not cognisant. Epileptic vertigo, moreover, frequently alternates with more or less complete epileptic convulsions during which consciousness is lost. Its onset has no direct relation to exertion.

In tumour of the brain, as in stomach vertigo, there may be headache and vomiting associated with giddiness, but apart from the fact that vertigo is rarely the symptom complained of by the subjects of cerebral tumour, as it is in Menière's disease and stomach vertigo, the distinction between the two conditions is readily made by the absence of symptoms of indigestion of food, which always accompany in greater or less degree stomach vertigo. Vertigo is also a symptom of tabes dorsalis and of ataxic paraplegia. Besides these functional and organic diseases of the nervous system, vertigo is not uncommonly associated with certain errors of refraction of the eye, of which the most important are myopia and hypermetropia. The attacks of giddiness in these cases are slight and are induced by the weakening of the ocular muscles (muscular asthenopia).

The vertigo which is of vascular origin is somewhat various in its causation. The prime cause of its occurrence is probably a deficient or irregular blood supply to the brain, which may be sudden. It is thus particularly associated with anæmic conditions: chlorosis, the profound anæmias, and anæmia from hæmorrhage, whether rectal, uterine, gastric, or pulmonary. It is associated also with two forms of cardiac disease, aortic regurgitation and fatty degeneration of the cardiac muscle, and with chronic Bright's disease, especially the granular contracted kidney with high arterial tension.

In the case of anæmia and of the cardiac diseases mentioned, the giddiness is associated with low arterial tension, and the attacks are usually induced by some exertion, either by some sudden movement such as sitting up in bed or some sudden effort during walking. Of these conditions chlorosis, and even to a greater extent aortic regurgitation and fatty heart, are closely associated with attacks of vertigo.

In many cases of chronic Bright's disease, the vertigo is associated with high arterial tension, which results in an irregular supply of blood to the tissues. Vertigo in Bright's disease has, like stomach vertigo, been ascribed to the circulation of poisons acting on the brain. But there is no direct evidence of this, and it seems most probable that the attacks are purely vascular in origin and not toxic.

There is, however, a form of vertigo which is induced by drugs, the chief of which are quinine, the salicylates, and strong iron preparations when given in some cases of chlorosis. In all these instances the vertigo is associated with headache and with buzzing in the ears, and is relieved on withholding the drug.

It cannot be too clearly stated that in the diagnosis of stomach vertigo, the more serious causes of vertigo must first be eliminated. In ordinary practice, an examination readily eliminates the different forms of anæmia, as well as diseases of the eye, aortic regurgitation, and chronic Bright's disease, while more difficulty will be found in eliminating Menière's disease and epilepsy. In the former, however, vertigo is often determinate in character, and is associated with deafness and with buzzing in one ear; in the latter the giddiness is associated either with unconsciousness or with unconscious acts, and is thus distinguished from stomach vertigo.

But little need be said of the other nervous phenomena associated with chronic indigestion of food. Buzzing in the ear is often observed with vertigo, and calls for no particular comment.

Inability to do mental work is often a subject of complaint, and is due partly to the continued irritation of the slowly digesting and undigested food in the stomach and partly to the malnutrition produced by the indigestion. In this case, the relation of work to meals and of the habits of the individual—whether sedentary or active—are of great importance.

As regards affections of motion and sensation, a tired feeling is a very common symptom of chronic indigestion of food, as well as a feeling of the legs giving way. There may also be cramps of the arms or legs and of the abdominal muscles. Cramps are not uncommonly observed in children after the eating of indigestible food, and may be described as a stomach reflex; but they are also seen in adults either from dietetic indiscretions in the healthy, or more often in the delicate, and in those convalescent from acute febrile diseases.

Sensation may also be affected, as shown in the tingling and numbness not uncommonly complained of, as well as a sensation of heat (without flushing) not only in the face but over the whole body.

It is difficult to say how far these symptoms are directly due to a disordered stomach, and how far to a general condition of malnutrition induced by the dyspepsia or not; but it is certain that in many cases they are relieved by treatment directed to the relief of the disordered digestion.

CHAPTER VII.

FUNCTIONAL DISORDERS OF THE STOMACH.

In the last chapter, the symptoms referable to indigestion of the stomach have been discussed in relation to their causation. It is evident from that and the previous chapters that functional disorders of the stomach cannot be grouped together as one disease or as two or more diseases, such as "acid dyspepsia," "atonic dyspepsia," "flatulent dyspepsia," etc. Such names as these only indicate a symptom, and if used are apt to mislead, since it might be thought, for example, that acidity was the only condition to be treated in acid dyspepsia, or that atony of the stomach the only condition to be dealt with in "atonic dyspepsia," flatulence the only symptom in "flatulent dyspepsia," etc. Functional disorders of the stomach cannot be scientifically or even practically grouped in this manner. It has been shown that in these disorders there are not only irregularities in the secretion of the gastric juice, but in the movements of the organ and in absorption, and, not least, varying conditions of blood supply during digestion as well as a temporary congestion and altered states of innervation of the organ. It is the object of the practitioner in each particular case to determine, as far as possible, what particular function or functions of the stomach are disordered, to ensure the success of the treatment adopted.

It is from all points of view essential to correctly estimate the symptoms and degree of functional disorders of the stomach, for on the one hand they complicate a large number of diseased conditions of the body generally, and on the other they are present in organic diseases of the stomach itself, viz. ulcer, cancer, and catarrh.

What may be called primary functional disorders of the stomach are those in which catarrh of the organ, an ulcer, and carcinoma are absent. They form by far the greater proportion of diseased conditions of the stomach, and are of great importance, since they cause a disturbance of the bodily functions which is curable, but which may lead to permanent mischief if attention is not paid to them.

Clinically, two groups of functional disorders of the stomach may be distinguished. In one, *gastric irritation*, the symptoms and the examination of the process of digestion and of the stomach show irritation of the organ, an irritation in which not only the secretion of the gastric juice is affected, but also the motor activity of the organ and the blood supply and innervation. This group contains the largest number of cases of functional disorder of the organ. The second group, *gastric insufficiency*, is one in which the functions of the stomach are deficient in activity owing to some general condition of the body, whether induced by a specific disorder or by the mode of life. While the first group of cases may be both primary and secondary (though usually primary), in gastric insufficiency the disorder is secondary, and includes cases usually classed as atonic dyspepsia. To gastric insufficiency may be added the signs of gastric irritation, since a stomach whose functions are deficient may still be irritated by the conditions to be discussed presently. A case which is primarily gastric insufficiency may thus become eventually one of gastric irritation. In both gastric irritation and gastric insufficiency catarrh may occur, but gastric irritation is the more closely allied to catarrh, which not infrequently shows a first stage of hyperacidity. A correct appreciation therefore, as far as possible, of the clinical conditions of gastric irritation and gastric insufficiency is essential before a study of the organic diseases of the stomach, gastritis, ulcer, and cancer, can be made.

Although, for practical purposes, it is convenient to divide functional disorders into gastric irritation and gastric insufficiency, it may be said that nearly all cases of indigestion of food—dyspepsia—show the symptoms of irritation of the organ, which is in many instances predisposed to by a condition of gastric insufficiency.

1. Gastric Irritation.

Gastric irritation may be defined as a functional disorder of the stomach in which there is irritation of the organ, produced by the food itself in the form in which it is swallowed or by the food in the process of digestion; it may be primary (*i.e.* occur in healthy individuals) or it may be associated with or predisposed to by certain diseases or general conditions of the body. Food bears to gastric irritation and to gastric catarrh the same relation that irritating particles and gases bear to bronchial irritation, bronchial catarrh and bronchitis, with the exception that food being necessarily taken by way of the stomach, acts as a continuous irritant, whereas the irritation of the bronchial tubes is intermittent. Both in gastric irritation and catarrh and in bronchial catarrh the condition may be predisposed to either by a local alteration of the organ (stomach or lungs), or by a general condition of the body. Similar observations may be applied to intestinal irritation and catarrh, and irritation and catarrh of any mucous membrane, and the resemblance between these conditions is not without import regarding the treatment of stomach affections, allowance being made for the differences in function.

Gastric irritation includes what is usually described as "acid dyspepsia," but this latter term by no means includes all the cases of gastric irritation, and is not a correct designation of the numerous cases of the disorder in which the motor activity of the organ is affected, as well as the secretory function.

Etiology.—The chief points in the etiology of gastric irritation have been discussed in the previous chapters, but it is important to review them from another and a purely clinical aspect.

Age and Sex.—Gastric irritation may occur at any age: in the infant, whether fed by the wet nurse or by the bottle; in the child, in adult life, and in old age. Sex has but little influence; in infancy and childhood the feeding is the same for both sexes, but in adult life, although sex still has but little effect, yet the mode of life diverges, the woman tending to an excess of food accessories (especially tea) and the man to an excess of food as well as food accessories (especially alcohol). At the beginning of life, gastric irritation is seen in some of its acutest forms, notably in infants brought up by the bottle. In middle age again, both in men and women, gastric irritation frequently starts. In women at the menopause, the first symptoms of dyspepsia may be noted, passing off as the menstrual change disappears. In young adult life, gastric irritation is common in both sexes, and its occurrence is explicable by factors in the etiology of the condition other than age.

Temperament plays some part in the causation of gastric irritation. Apart from any association of actual disease, those individuals with an excitable nervous system, who are usually described as of a "nervous temperament," are especially prone to gastric irritation. Some of the cases occurring in such patients may aptly be described as "nervous dyspepsia"; others again illustrate in a minor degree the influence of the nervous system over digestive processes. It is not uncommon, for example, in non-dyspeptic individuals for a period of worry, anxiety, excitement or disturbance to start an attack of indigestion; and in those who have previously suffered an attack or attacks of indigestion, and who can preserve themselves free from dyspeptic symptoms by care in diet and regulation of the mode of life, a sudden emotion of joy or sorrow or some other disturbing influence on the nervous system, may bring on an attack of indigestion.

Heredity.—It is always of great difficulty to determine the part which heredity plays in disease, except in such clear transmissions as deformities, mental peculiarities, and in such a disease as syphilis, where the actual disease is manifested in the infant soon after birth. It is always to be borne in mind that the same disease or diseased condition present in the parents and arising in the offspring may be the result of the mode of life or of exposure to the same surrounding conditions, whether of infection or not. In functional disorders of the stomach, heredity plays some part, although only a small one; but the question is not an easy one to settle or to discuss. It is the firm belief of many that they inherited a "weak digestion" from their parents. This is possible. Still it must not be forgotten that in families the children lead the same life as their parents; that they inherit robustness or general bodily weakness, or, as it may be expressed, increased or diminished resistance to disease. It is this inheritance of a general bodily condition which alone appears to predispose to functional disorders of the stomach. The inheritance of gout may also be a factor in gastric irritation. Gout is so frequently associated with stomach disorder (chiefly from the mode of living), that it has to be considered in relation to the inheritance of a "weak digestion." It is, however, by no means the greater number of cases of gout which transmit a tendency to functional disorder of the stomach, and in the majority of cases this is induced by the mode of life and by the food and food accessories. The same remarks would apply to the inheritance of the nervous temperament which is constantly transmitted from the parents to one or more of the children.

Climate and race have no particular influence on the causation of gastric irritation, except as regards the change in the mode of living and in the character of the food eaten. But frequently functional disorders are initiated by the sudden change from one climate to another, as from the temperate to the tropical regions, or even from the country to the town. Here, again, no particular effect can be ascribed to the climate, but only to a change in the mode of living and the diet.

Mode of Living—Occupation.—For a healthy existence a sufficiency of food and of exercise, and a congenial occupation are necessary. An insufficiency of food, even with much exercise, may lead to gastric derangement. But a more frequent cause is an insufficiency of exercise. It is those who lead a sedentary life who are subject in the greatest degree to gastric irritation. A sedentary life means not only very little exercise, but it means in many instances the partaking of large meals often with a large quantity of food accessories; in men of alcohol, in women of tea. Again, even when there is a sufficiency of food and a fair amount of bodily activity, such as walking, gastric irritation arises, owing not only to irregularities in diet, but also to the fact that exercise to be really beneficial must be of interest, and not merely of necessity; it must be recreative, and not only on business. Gastric irritation induced by these conditions is seen not only in the well-to-do, but in the poorer classes; in those of the learned professions who lead a sedentary life, and in the seamstress who works for ten hours a day in a badly ventilated room by gaslight. The change from an active to a sedentary pursuit is often the beginning of gastric irritation, especially in middle age when the desire for active exercise tends to diminish.

Arrangement of Meals.—The question of the distribution of meals during the day has already been partly discussed in the preceding pages (Chapters II. and III.). The principles on which meals ought to be regulated are very simple, viz. that a large meal ought to be followed by a period of bodily rest and recreation (not sleep), and should not be succeeded by another large meal within five hours. Meals habitually taken hurriedly lead to indigestion of food; so also does work, mental or bodily, after a heavy meal. Undoubtedly the most rational arrangement of meals is a large breakfast, a moderately large dinner in the middle of the day, and a smaller meal in the evening; but in the exigencies of modern life this arrangement is impossible, as the midday meal has to be followed by work. In the matter of meals each individual soon finds what is best for his digestion. One point must be emphasised, viz., that the partaking of a meal of any size one or two hours before going to bed is very prejudicial to good digestion, and if the time of retiring is about eleven, no meal ought to be taken after 7 P.M.

Food and Food Accessories.—Of all the factors in the etiology of gastric irritation, food and food accessories are the most potent. This has been fully discussed in Chapter III. (p. 56 *et seq.*). Food indeed is the direct agent in the production of all cases of gastric irritation: food from its bulk, its indigestibility, and from its irritant or other properties (*loc. cit.*).

A meal is taken by a healthy person, which from its nature is not digested before the next meal; this is again a large meal, and is succeeded in the evening by another large meal, so that the stomach is never at rest. In time the meals are not digested, and gastric irritation is the result. Sufficient has been said in the previous chapters to explain this fully. The effect is still greater if any general condition predisposing to gastric irritation is already present. Food accessories play as great a part as food in the production of gastric irritation, or even greater; their physiological effect is one which delays the chemical processes of digestion in the stomach (Chapter II.); but they are also irritants, so that alcoholic drinks, tea, spices, and highly-flavoured sauces rank among the most potent exciters of gastric disturbance. Two chief exciting agents of this class are alcoholic drinks and tea taken in excess, or in an irregular manner. As regards alcoholic drinks, even when not taken in excess, beer may be a cause of gastric irritation, as well as the light and heavy wines. This is to a less extent the case with the ardent spirits, taken moderately with meals in a diluted form. Alcoholic excess is, however, very common, not only to the extent of habitual tipsiness and drunkenness, but to the extent which is excess for the individual. The digestion in one person may, for example, be affected by an amount of alcohol which would do no harm whatever to another. The taking of alcoholic drinks between meals has a greater effect in the production of gastric irritation than when drinking is confined to the meal-time. A distinction must be made between alcoholic drinks and alcohol, reckoning the well-watered ardent spirits (whisky, brandy, gin) as alcohol. Alcoholic drinks, such as beer and wine, have a greater effect in retarding digestion than pure alcohol itself (Chapter II.), probably from the organic and inorganic salts they contain; they are therefore more harmful to "weak" digestions. Alcohol itself is an irritant to the stomach, and is a cause of acidity, as has been previously discussed in detail (Chapter II.).

Tea is a common factor in the causation of gastric irritation: strong tea frequently drunk during the day. It is actually a poison to many individuals with a weak digestion. It acts not only by retarding digestion, but as an irritant, owing to the tannin it contains. Coffee and cocoa made with milk are not so irritating.

Starvation or insufficiency of food is in some cases a cause of functional disorder of the stomach. Amongst the poorer classes, such cases are sometimes seen; as when following a period perhaps of months of great insufficiency of food there is digestive disturbance, which is chiefly shown by the fact of food acting as an irritant, and being undigested and ejected.

Relation of Gastric Irritation to other Diseases.—Gastric irritation may arise in ulcer or in cancer of the stomach. Ulcer and cancer in the early stage must be looked upon as localised diseases of the organ, not at first affecting its functions of secreting a proper amount of gastric juice in response to the stimulus of food, and not to any great extent affecting the motor power of the organ. Soon, however, signs of gastric irritation may arise in both diseases, and may mask the symptoms proper to the disease.

Other diseases bear an important relation to gastric irritation (Chapter IV.). They all tend to produce a diminished activity of the functions of the stomach, and therefore the first condition is one which would be classed under the heading of gastric insufficiency. Still, gastric irritation often supervenes, and has much the same characters as when primarily produced. Of non-infective disorders, chlorosis and chronic Bright's disease are those most favourable to gastric irritation; cardiac valvular disease to a less extent. In chlorosis, as well as in Bright's disease, probably all the functions of the organ are diminished in activity (see Gastric Insufficiency, p. 211).

Among infective disorders, *tuberculosis,* especially of the lungs, is most favourable to the development of functional disorder of the stomach (p. 90). The symptoms of gastric disturbance may precede the development of the physical signs of the disease; and it is not improbable that, in some cases, the presence of gastric disorder may predispose to the development of tuberculosis. But in most cases, the gastric disorder develops in the course of the pulmonary tuberculosis, and is not infrequently of a severe character. There are several features of pulmonary tuberculosis which are predisposing to the development of gastric irritation. The anæmia produced, the exhausting fever, the debilitating night-sweats all tend to produce a diminution of activity of the organs.

among which the stomach suffers, and is thus liable to be irritated by the food taken. The swallowing of sputum may also act as an irritant as well as excessive cough. On the other hand, there are very numerous cases of pulmonary tuberculosis in which no stomach symptoms arise, even in the last stages of the disease.

In *chronic malaria* the stomach may become disordered, but it is chiefly in the *convalescent stages of certain acute diseases* that gastric irritation is apt to be induced, as well as catarrh of the organ. During convalescence, when the appetite is returning, injudicious feeding may start a functional disturbance; slight at first, but increasing and lasting for years. This may occur in convalescence from typhoid fever, scarlet fever, measles, rheumatic fever, and even cholera. In such diseases, during the acute stages, the secretory and motor activity of the stomach is diminished, and it takes some time for the organ to recover its normal condition. Food in too great bulk or of an indigestible character readily acts as an irritant and produces the symptoms of gastric irritation.

In summing up the etiology of gastric irritation, it is clear that food and food accessories are the direct causes of the functional disturbance in association with the mode of life. These may be the primary and only causes, but they are aided by other factors, such as the gastric insufficiency of some general diseases and of the convalescent stages of certain infective diseases.

Symptoms.—The symptoms produced by gastric irritation are in the majority of cases quite simple, but in a certain number are complex, owing to the effect of the stomach condition on the nervous system.

Stages of the Disorder.—Gastric irritation may be considered as having two stages, in each of which the diseased condition may show itself for a long period of time without anatomical changes in the mucous membrane occurring.

In the first stage the pathological conditions present are:—

1. A varying degree of congestion and of nervous irritability of the organ.
2. An increased and prolonged acidity of the gastric con-

tents, due chiefly to an increased secretion of the acid gastric juice, but also to the organic acids of the food.

3. A delay of food in the organ, owing to the weakness of the muscular walls (atony). Moderate dilatation of the organ is not infrequently present.

In the second stage the overworked and irritated organ shows a diminution of function by—

1. A diminished secretion of the gastric juice, especially of the hydrochloric acid.

2. A greatly diminished motor activity whereby the delay of food is greatly increased, terminating in dilatation of the organ.

The first stage is, therefore, characterised by hyperacidity, due to hydrochloric acid and by a varying degree of weakness of the muscular walls; the second stage is similar to the cases to be described as gastric insufficiency. In both stages there may be nervous irritability of the stomach.

Mode of Onset.—The modes of commencement of a chronic disease are important to elucidate. Generally speaking, the recognition of a fully developed diseased condition is not difficult, but the diagnosis of disease in its early stages often presents great difficulties. Inasmuch as in diseases of the stomach, functional disorder is frequently found in association with organic disease, as well as existing by itself, it is important to study the origin of functional disorder in order to distinguish it from organic disease. Taking the average of cases, it may be said that clinically organic disease differs from functional disorder in the fact that there is a definite period known to the patient during which the symptoms have lasted, whereas functional disorder is insidious and gradual in onset, and when its origin is sudden there is some definite cause determining it.

Functional disorder of the stomach may have two modes of origin—a definite or an insidious.

1. When its origin is definite, it is often acute. By a definite origin is meant the occurrence of the diseased condition after some pathological event in the patient's history. Thus

gastric irritation not infrequently dates from the period of convalescence of the patient from an acute febrile illness, typhoid fever, measles, scarlet fever, rheumatic fever, less commonly of pneumonia. In such cases, as has been explained, during the febrile stages, there is a diminished functional activity of the stomach, both secretory and motor, and during recovery indiscretions in diet produce gastric irritation which not infrequently persists for a long period. The stomach in such cases is not allowed to recover owing to the irritant action of unsuitable food. As will be seen, also, catarrh of the organ frequently has a similar origin in the convalescent stage of acute diseases. Gastric irritation may, on the other hand, be acute in origin, due to the action of a strong irritant, and the acute gastric irritation may become chronic. Irritant poisons from the effects of which the patients recover are frequently starting-points of gastric irritation, but there is, as a rule, more than functional disorder in those cases, as there is actual destruction of the gland tissue, or there may be a chronic catarrh (see Gastritis Toxica). Irritant food may be the starting-point, more particularly tainted food and high game, the acute irritant symptoms being produced by the products of bacterial decomposition of the food. One of the commonest modes in which gastric irritation occurs acutely is the sequence of recurrent attacks of acute gastric irritation, due to intemperance in food and food accessories (alcohol and tea). Lastly, gastric irritation may have a definite result owing to a change in the mode of life.

Insidious Onset of Gastric Irritation.—Although gastric irritation may arise acutely in the above conditions, yet in the majority of cases its onset is insidious. Repeated slight indiscretions in diet, whether in respect of the food or food accessories, or in the regularity of the mode of living, although apparently only temporary in their effect, may commence a period of pronounced functional disorder of the stomach. At first an occasional indiscretion in the diet does no harm; a temporary fulness or discomfort after a meal soon passes off and no attention is paid to it, but unless the diet be regulated with care the symptoms recur and increase in intensity. Such indiscretions of diet do not always act alone: they may do so, *i.e.* they may in a quite healthy individual lead to functional disorder, but they are commonly combined with other conditions which predispose to the gastric disturbance. Such conditions are a period of worry, or of sedentary work, of work in a close heated atmosphere, of change of diet as in moving from one country to another, or of the mode of living, as in moving from the country to the town or *vice versâ*.

Symptoms and Course of Gastric Irritation.—In gastric irritation there is a varying congestion of the organ dependent on the presence of the food, so that there is not that normal response on the part of the vessels to the stimulus of food which is necessary for the continuance of normal digestion. This varying congestion passes off as the stomach becomes emptied or the irritant portions of the meal are expelled. In the second place, there may be increased irritability of the nervous mechanism of the organ, which may show itself on the side of increased secretion or of decreased motor activity, both being induced by food. The increased activity produced in this way is a feature of gastric irritation when developed. The actual irritants, therefore, as it has already been fully explained, are the food, the food accessories (especially alcohol, tea, spices), and the increased acidity of the gastric contents due to hydrochloric acid. There may be an increased secretion of hydrochloric acid with a diminished motor activity; or spasm of the organ may occur; in some cases there is a deficient secretion of hydrochloric acid.

Acute Gastric Irritation.—Acute gastric irritation occurs, as previously explained (p. 65), simply as the result of large meals or of irritating food. The symptoms in such a case are a sense of fulness and discomfort occurring two or three hours after the meal, followed it may be by very acid eructations of small quantities of the stomach contents and of gas, accompanied by nausea, and frequently terminating in the ejection of the whole contents of the organ. Relief to the symptoms is then obtained; but for some time the organ is incapable of doing much work, so that for a period of twenty-four hours there is loss of appetite, and some slight discomfort beginning in half an hour or an hour after food and lasting about an hour; flatulence and nausea with occasional acid eructations may also be present. The irritated stomach then recovers itself, and ordinary food can be taken without discomfort. Actual pain may be felt in the stomach region, the presence of pain depending on the degree of irritation and on the condition of the nervous system. Pain, for example, is a well-marked symptom in severe cases where high game or tainted food causes gastric irritation, as well as in cases of irritant poisoning. Again, in individuals with an irritable nervous system, pain in the epigastrium is frequently a well-marked symptom. In ordinary cases, however, when food and food accessories cause acute gastric irritation, pain is not infrequently absent.

Another result which may, in rare instances, follow the ingestion of a large meal is acute dilatation of the organ, practically a paralysis of the muscular coat (see Dilatation of the Stomach, Chapter XIV.).

Physical Signs in Acute Gastric Irritation.—During the time that the irritant food remains in the organ, the stomach is in some instances firmly contracted around it, in others it is moderately dilated. The first condition is shown by a prominence of the epigastric region; and on palpation, by the sensation of a rounded mass with ill-defined edges. Dilatation is shown by the percussion note (tympanitic) extending downwards towards the umbilicus and into the left axillary region as high as the fourth rib. The upper region of the abdomen is in these cases often flaccid, and splashing of the stomach contents may be obtained on succussion. In the majority of cases there is little or no deep tenderness over the stomach region, but manipulation not infrequently causes a slight eructation of gas or of the stomach contents, and it may even excite vomiting. A diffuse tenderness, of varying degree, is observed only in those cases where the irritation is great. In the period following the emptying of the stomach, whether by vomiting or by passage of the contents into the duodenum, the stomach may remain moderately dilated.

Symptoms not Referable to the Stomach.—Acute gastric irritation produces a thickly coated tongue, yellow or white, and a lingering, nasty and nauseous taste in the mouth. The bowels are usually opened, and a loose, frequently copious motion is passed once or twice in the twenty-four hours. There is a disinclination to exertion, mental or bodily, although exercise commonly alleviates the symptoms. There is headache, which is to be ascribed to the character and quantity of the alcoholic drinks taken rather than to the food itself. The complexion is pale, somewhat yellowish and "washed out."

These are the symptoms of what may be described as a "food debauch" or a "bilious" attack, and they are of importance as being explanatory of the exacerbations in chronic gastric irritation which are so common. The repetition of "food debauches" is a frequent cause of chronic dyspepsia.

Chronic Gastric Irritation.—In chronic gastric irritation there is prolonged functional disorder of the stomach, in which there may be (and usually are) remissions of greater or less duration, and in which there are commonly acute or subacute exacerbations. The symptoms are referable to the food and food accessories taken, and are divisible into three stages according to the degree of irritation: the first stage may be described as one of discomfort of digestion, and leads to the second, in which the discomfort is more marked, and is associated with hyperacidity of the stomach contents and great irritability of the organ. The third stage is one in which the functions of the organ are diminished, although the signs of irritation are not absent. The symptoms of these stages overlap to a great extent; and, moreover, in each stage one or more symptoms may obtain prominence and give quite a special feature to the individual case. In the first stage, for example, the symptoms of the gastric disturbance may be slight, and the prominent symptoms be confined either to the circulatory or to the nervous systems. In the second stage, acid eructations and flatulence are the chief symptoms, although atony of the muscular coat is a prominent sign in some cases, while in the third stage, the symptoms of diminished functional activity are shown in diminished secretion and in dilatation of the organ, which may mask all the other symptoms. It is, however, important to note that in each individual case of gastric irritation, the features of the case as shown in the symptoms maintain a certain type.

The symptoms of gastric irritation, in whatever way it arises, may be divided into two classes, general and those referable to the stomach. The latter are shown in a sense of fulness, weight, and oppression after eating, in pain and oppression in the chest and between the shoulders; and by occasional flatulence and acid eructations. The more general symptoms are those chiefly referable to the nervous system (Chapter VI.). They are headache, palpitation, drowsiness, mental depression, sleeplessness, and the other symptoms already described.

The symptoms referable to the stomach always appear in relation to food, coming on at various times after the meal, and are usually most marked after the principal meal of the day, or in the evening, when the stomach has had to cope with at least three meals during the day.

The sense of fulness, weight, and oppression in the epigastric region may come on directly after eating, but is often delayed for half an hour, or for one or two hours; it may then last four or five hours or until the next meal. Pain in the chest and between the shoulders bears a similar relation to the meal. The delay of most of the symptoms is ascribable to the fact that the digesting food is most irritating to the organ during the period of greatest acidity. In about half an hour the amount of hydrochloric acid in the normal stomach contents is appreciable, and the percentage then rises to a maximum in two to four hours according to the size of the meal, and then rapidly declines as the stomach is emptied. In gastric irritation the secretion of acid is more rapid, and the degree of acidity one hour after a meal may exceed the normal. Part of the distress is ascribable to the continued peristalsis of the organ necessary for digestion, and part to the long delay of acid food in the organ. The symptoms mentioned may, however, be observed in cases of hyperacidity and rapid digestion; and the degree of acidity of the gastric contents must be considered as their chief cause. It is noticeable that whereas both the sense of pain and oppression in the chest and the sensations in the epigastrium are described at first by the patient as occurring after a meal, in prolonged cases the relation to food is not observed owing to the fact that the stomach is always irritated by the presence of food in a more or less digested condition, the organ in fact never being empty even after a night's rest from food in some cases. Occasional flatulence is observed after a meal, or in the evening, and sometimes the symptom has reference to special articles of diet which disagree (viz. tea, carbohydrate food, etc.). Acid eructations are sometimes complained of, but vomiting is very occasional.

In the later stages of gastric irritation, the chief stomach conditions present are hyperacidity of the contents due to an excessive secretion of hydrochloric acid in the gastric juice and a tendency to weakness of the muscular coat, leading to dilatation of the organ. Although the symptoms preserve the same type as those just described, they develop in certain directions, viz. acid eructations with flatulence are more frequent, vomiting is more frequent, the eating of food becoming a source of fear to the patient. There is no particular feature of the acid eructations; they occur, like the pain and discomfort, in from half an hour to two hours after a meal, and are due to a regurgitant action of the stomach when the acid digesting food touches the cardia.

Flatulence may be excessive, and is apt to be greatest towards the end of the day; at first coming on after a meal, it soon apparently loses its relation to meals.

The character of the vomiting needs more particular mention. The vomiting in this case is due to the presence of irritating food in the organ, and is dependent on the condition of the nervous system. If there is great irritability of the nerves of the stomach, whether as the result of long continued gastric irritation, or of a general irritability of the nervous system, such as occurs in "nervous" individuals and in some anæmic subjects, in which a slight peripheral irritation produces a great effect, vomiting is readily excited by the presence of food in the stomach. In such cases vomiting may occur after every meal, directly after the meal, but more commonly it comes on in from one to two hours afterwards. Patients suffering from gastric irritation are subject to such attacks; but they are only attacks, readily disappearing under treatment. A not uncommon history is that the patient has been the subject of indigestion of food for a long period—one, two, or more years, that the distress has been increasing during the last few months, and that for a period perhaps of one month there has been vomiting directly after meals or after a period of two to four hours. In other cases vomiting may occur in fourteen days or a month after the onset of the gastric irritation, and this is especially so in cases in which there is a predisposition to gastric disorder owing to some general disease; more particularly is such a rapid development of symptoms noticed in chlorosis and in convalescence from acute diseases. In still other cases the vomiting is only occasional, and due to some special indiscretion of diet, a very large meal or an excessive amount of alcohol or tea, while in many cases no vomiting occurs. As a rule, however, occasional vomiting is present, if there is well marked hyperacidity of the stomach contents during digestion for a long period.

Character of the Vomited Matters.—This has already been fully discussed (Chapter IV. p. 81, Chapter V. p. 142). There is no mucus in the vomit, which consists chiefly of digesting food, and shows the presence of peptones by the ordinary tests. The vomit is very acid, the acidity is due mainly to hydrochloric acid, which even in a short time after the meal may be in much higher proportion than normal, as high as over 0·3 gramme per cent. In some cases the acidity of the vomit is within the normal limits. Pepsin is present, and usually in a very active condition.

Some cases of hyperacidity of the gastric contents are rather different to the more common ones just described. They are characterised by rapid digestion of the food and rapid emptying of the stomach; they are indeed cases of hyperacidity with irritability of the muscular coat of the organ. There is in these cases the same distress in the chest and discomfort during digestion which have been described, but soon after food a sense of emptiness is felt, which is not relieved by more food. Indeed in some cases, the appetite returns soon after a meal has been taken.

Cases of gastric irritation may continue in the stages described for a long period—many years; they have remissions of greater or less duration, and exacerbations with the symptoms described, rendering the sufferer's life intolerable for a time, but relief is soon obtained, as the patient has to undergo treatment. Complete recovery is frequent, but on the other hand, through neglect or other causes, permanent injury may be done to the organ, whereby the digestion is always "weak," or a severe injury may result which requires months of treatment to remedy. The stomach irritated in the manner described may become inflamed, catarrh being produced, or its functions diminish in activity without inflammation occurring. The first diminution is seen in the motor activity of the organ, so that the delay of food is increased and the organ tends to dilate. This dilatation is only a moderate one, and rapidly diminishes under treatment; it is frequently kept up by an irregular and unsuitable diet. As an illustration of this condition, the following case may be quoted:—A woman, aged forty years, had suffered from attacks of indigestion of food, "off and on," for several years; she came complaining of symptoms which had been present more or less for one or two years. These consisted chiefly in pain and oppression in the chest after eating, coming on either directly or in one hour after food. The appetite was bad, and the bowels were only opened when an aperient was taken. Vomiting after every meal had of late occurred, the vomit being described as frothy, watery, and sour. This was then a case of prolonged gastric irritation which came under treatment during a subacute exacerbation. On examination, no organic disease was discovered in any part of the body, but the stomach was dilated. By percussion it extended as far downwards as the umbilicus, to the right nearly as far as the right nipple line, and in the left axillary region as far upwards as the fifth rib: there was no tenderness over the stomach region. All the symptoms diminished rapidly under treatment. In such cases dilatation of the organ is more apt to occur between the ages of forty or fifty than in young adult life, except where the gastric irritation arises in the course of chlorosis or during the convalescent stage of acute febrile diseases; in these cases dilatation frequently comes on very rapidly after the onset of the functional disorder (see Gastric Insufficiency).

In the later stages of gastric irritation there may be extreme dilatation of the stomach with the symptoms referable to that organ. In most such cases the secretion of gastric juice is insufficient. Their history shows that there has been a long period of "dyspepsia," which has ended in the present condition; the dyspepsia presenting the features described as characteristic of gastric irritation. Besides the physical signs of a dilated stomach (Chapter XIV.), the symptoms shown by such patients are the following—flatulence, pain, and vomiting. The flatulence is often excessive. The pain is referred to the stomach region, and is noticeable as not continuous, but coming on at intervals. It is not related to a meal, but that it is due to the presence of food, digested or partially digested, is shown by the fact that it precedes vomiting, which relieves it. Indeed, patients frequently excite vomiting in order to relieve the pain. The vomiting is also not related to meals, but occurs at long intervals; once daily towards the evening, but more frequently once every two or three days, when the large accumulation of fluid and of partially digested food with a little mucus is rejected by the stomach. In the intervals of pain there is no tenderness in the stomach region, but during the pain this symptom may be noticed. The vomit has the characters of fluid from a dilated stomach (*loc. cit.*). There is no hæmatemesis or melæna. Such patients are greatly relieved by treatment, but there is permanent injury to the organ, from which complete recovery is impossible. Permanent injury may be done to the organ without the case progressing so far as we have just described. Gastric irritation of long duration may be relieved by treatment, but owing to neglect of a proper regimen, the patient may always be subject to a recurrence of the disorder. Such patients can, however, by avoiding indiscretions in diet, lead a useful and comfortable existence. This permanent effect of gastric irritation may be associated in middle age with a greater or less degree of atrophy of the gastric glands.

There are other symptoms in gastric irritation besides those more directly referable to the stomach.

Appetite.—In the early stages the appetite may be normal or even increased, and may sometimes be described as voracious. In cases of hyperacidity of the stomach contents due to hydrochloric acid, the appetite is not diminished. In prolonged cases of the disorder the appetite is apt to become capricious, varying from time to time, and preserved only for dainty articles of diet. Where there are evidences of greatly deficient motor activity of the stomach, the appetite is almost invariably diminished.

Mouth and Tongue.—The tongue is frequently coated with a white or yellow fur, especially in the mornings on waking. It may, however, be clean, especially during the day. On waking, too, there is constantly a nasty taste in the mouth, which may contain a clammy mucus. The taste is nauseating, sometimes bitter, and with the furred morning tongue, is no doubt to be ascribed to eructations of irritating gas or liquid from the stomach during the night. These symptoms mostly occur in those who take late meals, which are very slowly digested during sleep, or in those in which there is great delay of food in the organ from whatever cause.

Salivation may occur, most frequently in cases of great hyperacidity of the stomach contents, and in the subacute exacerbations of gastric irritation; it usually occurs after meals, and during the period of distress in digestion. In prolonged cases, owing to the effect on the general system, the tongue may become pale and flabby, and tooth indented. In some cases the sense of taste is diminished or even lost; a symptom the origin of which it is difficult to explain. The sense is usually recovered.

Bowels.—In some cases the bowels are regularly opened each day. This is, however, not the rule; constipation usually being present. The bowels are opened once in two or three days, or may be opened only once a week. Apart from the fact that, especially in women, constipation is a habit which may be acquired by irregularity in going to the closet, gastric irritation tends to produce constipation. This is very evident in the subacute attacks which arise in the disorder.

Another condition of the bowels which is not infrequently present in the course of gastric irritation is an alternating condition of constipation and diarrhœa. In well marked constipation there may be diarrhœa: the colon may contain a large quantity of solid fæces, which may cause slight diarrhœa (the passing of small liquid stools) by irritating the gut. In gastric irritation the condition is not always of this nature; on one or two days one or more liquid or semi-solid dark stools may be passed, and on the following days there is constipation. It is difficult to exactly determine in what conditions this alternating diarrhœa and constipation occurs; but two of the conditions are the continued eating of large meals consisting largely of meat, and a sudden change of diet. The diarrhœa just considered has no direct relation to the eating of a meal. A third condition of the bowels in gastric irritation is seen in the onset of diarrhœa directly or soon after a meal—"lienteric diarrhœa." There are many conditions other than gastric irritation in which lienteric diarrhœa may occur, some of which have to do with indigestion of food in the small intestine, while others are associated almost solely with disordered innervation of the intestinal tract. But in certain cases of gastric irritation, the food appears to be rapidly discharged from the stomach and a rapid peristalsis of the intestinal tract is initiated. That the lienteric diarrhœa is to be ascribed to the gastric irritation is shown by the fact that in the course of the case it comes on in attacks lasting from one to three weeks, and that it is relieved by treating the stomach condition. The change seems primarily to be one of increased motor excitability of the organ. Besides a meal, hot drinks will excite the diarrhœa, which is shown in the passage of watery stools, containing in many instances undigested food. It is observed chiefly in children and women, but also in men; and unassociated with gastric disorder, it complicates chronic febrile diseases, *e.g.* pulmonary tuberculosis. The passing of a liquid stool directly after a meal is soon succeeded by the passing of two or three stools after meals; and in some of these later cases the patient simply states that diarrhœa is present, but does not directly connect them with meals, unless questioned on the point.

Condition of the Urine.—A small quantity of high-coloured urine is not infrequently passed during and after a subacute attack of gastric irritation. In the chronic disorder, the chief changes that occur in the urine are a tendency to alkalinity of the urine, a diminution in the quantity excreted and an excessive excretion of phosphates.

Albuminuria only occurs as a result of the ingestion of particular articles of diet, such as white of egg: albumosuria may be found, more particularly when the organ is dilated.

Effect on General Nutrition.—Gastric irritation is not associated with any particular build of body; it may occur in the stout or the thin, in the well-nourished or in the badly-nourished. In many cases of the disorder there appears to be but little effect on the general condition of the body; the weight is maintained, and the strength; but a continuance of the disorder leads to general effects which may be profound. In the early stages, or even when gastric irritation is subacute and in the second stage, there is usually no obvious wasting of the body; but the face is paler than natural, and the bodily strength diminishes. Irritability of the muscles on tapping (myotatic irritability) is frequently observed, especially in individuals with an excitable nervous system. When, however, the gastric irritation has been much prolonged, more especially as the patient approaches middle age, and in women at the climacteric, there may be wasting. This is particularly so when there is dilatation of the organ, associated with the permanent defect in the digestive power which has already been discussed. The loss of flesh has, however, some characteristics. Although there may be some permanent diminution of weight during the course of three or four years, yet if the patient be weighed week by week his weight will be found to fluctuate greatly, increasing when the disorder is improved, decreasing on a recurrence of the symptoms. Progressive wasting, dating from a known period, occurs in organic disease of the organ. It is the exception even in the most prolonged cases of gastric irritation, and when it occurs it is due to some other cause than the stomach disorder. Indeed, patients in whom a disordered gastric digestion exists may actually gain in weight so as to become obese, especially towards the middle period of life.

Course and Prognosis.—The course of primary gastric irritation depends chiefly on the means adopted for its cure. It is very amenable to medicinal and dietetic treatment, and therefore the prognosis is good. But the usual course of gastric irritation is shown in recurrent attacks of greater or less severity, and these are almost solely due to the fact that after a period of relief from the symptoms of indigestion of food, the patient loses caution and commits irregularities in the mode of living and in the diet, which brings on another attack. If gastric irritation supervenes on gastric insufficiency, the immediate prognosis is good, since the disorder can be controlled by appropriate treatment; but the gastric insufficiency still remains, and may be very slow in recovery. In all cases where gastric irritation is secondary to or occurs in the course of organic disease elsewhere than in the stomach, the course of the disorder is prolonged, but it is still amenable to treatment.

Diagnosis.—The diagnosis has chiefly to be made from chronic catarrh (see Chapter VII.). For the diagnosis between functional and organic disease of the stomach, see pp. 262, 435.

Treatment.—The treatment of gastric irritation must be conducted on lines directed to procure rest for the stomach, to allay irritability of the organ, and to counteract hyperacidity.

Rest to the stomach is procured by attention to the diet (Chapter XI. p. 322) and by the use of sedatives (Chapter X. p. 300); irritability is also allayed by these means, and hyperacidity is temporarily relieved by antacids (Chapter X. p. 290), and cured by rest and by alteration in the diet.

Of great importance in the prevention of the recurrence of attacks of gastric irritation is attention to the condition of the teeth and to general hygienic regulations (Chapter XII.).

Gastric Irritation with Prominence of Nervous Symptoms: Nervous Dyspepsia, Neuroses of the Stomach.—In the majority of cases of gastric irritation there is a combination of symptoms which readily points to the kind of disorder present: the epigastric fulness, weight and discomfort following meals, the pain in the chest, the acid eructations and flatulence are characteristic. These symptoms may intermit or become more violent, subacute or acute gastric irritation occurring, in which there is constantly a great deal of food in the organ, with hyperacidity and prolonged acidity and occasional vomiting. In those comparatively rare cases in which gastric irritation leads to great and persistent dilatation of the organ, the symptoms of this with those of deficiency of function in the organ are manifest, and are directly referable to the stomach condition.

There are, however, many cases of gastric irritation, and indeed of other disorders of the organ (gastric insufficiency), in which certain symptoms attain such prominence as to give a feature to the particular case. Such symptoms are without exception referable to some change in the nervous system, and are by some included under the terms nervous dyspepsia and neuroses of the stomach. On the other hand, in certain nervous conditions (hysteria, neurasthenia) there are symptoms referable to the digestive organs, primarily produced by the nervous condition itself, but induced by the presence of food in the organ. The working of the nervous mechanism of the stomach in its effects on its functions is only imperfectly understood, and as far as our knowledge goes has already been discussed (p. 14). For the present purpose it is sufficient to remember that the nervous mechanism is an intricate one, and is partly local, but chiefly central, and that not only the higher centres but the special sense centres of sight and taste are connected with the nervous mechanism of the organ. In gastric irritation, the irritability of the nerves of the stomach is increased by the continued or intermittent peripheral irritation. So that in one direction gastric irritation may lead to increased reflex effects starting from the stomach, and excited by the presence of food itself or of hyperacidity. Connected with this there is an increased excitability of the central nervous system in response to a peripheral stimulus, which may be induced by the stomach condition or by some disorder or disease elsewhere than in the stomach. On the other hand, a disordered condition of the central nervous system leads in some cases to gastric symptoms, which may at first be purely nervous in origin, but which soon show the evidence of gastric irritation: such cases occur in hysteria, neurasthenia, and hypochondriasis, and in those conditions of the nervous system induced by overwork, worry, and like causes, or in the convalescent stage of acute diseases.

The nervous symptoms which may individually become prominent have been fully discussed in the last chapter. Thus headache, frontal, vertical, or occipital, with or without constipation, may be the feature in one case, and vertigo in another. Palpitation and flushing of the face, especially in women of middle age, may obscure the symptoms of indigestion in another case; and in yet other cases mental perversion, such as mental apathy and melancholia, may be the chief symptoms observable. Craving for extraordinary articles of diet or for articles absolutely uneatable (such as chalk, sawdust, etc.) is a sign of mental perversion and may be classed with *anorexia nervosa* or the loss of appetite due to a perverted nerve condition, in which there is profound wasting. These symptoms, however, may or may not be associated with functional disorder of the stomach.

In some of the cases similar to those under discussion, a prominent symptom is the eructation of gas and of the acid contents of the stomach, commencing soon after a meal and continuing during the whole time of digestion. This symptom, which may be called *eructatio nervosa,* is not necessarily associated with any chemical change in the process of digestion, but the liquid brought up may be watery, neutral, or even alkaline. In fact the chemical processes as determined by a test-meal may be normal; but there exists an irritability of the stomach which is shown by the partial voiding of the acid digesting food or of the small amount of liquid which remains in the organ after digestion. Indeed, in such cases vomiting is frequently observed, and may become the chief symptom in the disorder—*vomitus nervosa.* Both these symptoms occur almost solely in women, generally under forty years of age, and are usually associated with well-marked constipation. Thus in one particular case in a young woman aged twenty-four, the vomiting occurred after each meal for periods during three years. The appetite remained good, and there were no symptoms of indigestion of food.

Cases in which these nervous symptoms are prominent may be classed as "nervous dyspepsia," and Leube[1] would limit this term to cases in which the process of digestion was normal. This limitation, however, is not practically accurate, and it is best to consider these cases under two classes, viz.:—

1. Cases of gastric irritation with prominence of one or more nervous symptoms.

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[1] "Ueber nervöse Dyspepsie," *Deutsches Arch. f. klin. Med.*, Leipzig, 1879, vol. xxiii. p. 98.

2. Cases in which there is normal digestion, but there are nervous symptoms either general or related to the stomach, *e.g. eructatio* and *vomitus nervosa.*

The first class of cases has already been fully considered (Chapter VII.). The second class of cases occurs in those who are the subject of the numerous manifestations of hysteria or neurasthenia. As regards the stomach symptoms Leube's summary may be taken as practically accurate, viz., that nervous dyspepsia occurs in young adults (chiefly females) above the age of puberty, that the appetite varies, that there are numerous gastric reflexes, and that while the process of digestion is good, there are occasional attacks of vomiting and acid risings. There is no tumour, and the stomach is not usually dilated. One important point of distinction between such cases and cases of gastric irritation, chronic catarrh, and ulcer of the stomach is that they do not readily yield to treatment directed to relieve a stomach condition or to even general treatment. They are commonly most intractable, as severe as those of hysteria and neurasthenia, of which, indeed, they are local manifestations.

Course and Prognosis.—The course and prognosis of gastric irritation when it presents itself in the forms of "nervous dyspepsia" is widely different from that of simple irritation, inasmuch as the stomach condition is one of a series of symptoms of more or less profound functional disorder of the nervous system. Relapses are therefore common and much permanent benefit is not, as a rule, to be expected, although relief can be given to the most prominent symptoms.

Treatment.—This is twofold: a general tonic treatment, both medicinal and hygienic (baths, exercise, massage—Chapter XII.); and a local treatment to the stomach, consisting of sedatives (Chapter X.) to relieve irritability of the organ, and antacids to relieve hyperacidity when present. The dietetic treatment is to be prescribed on the same lines as those of simple gastric irritation, but in the cases under consideration, much harm will be done by reducing the diet too much or prescribing it on too rigid lines, because such patients will not infrequently be found able to digest articles of diet, which would be irritating and utterly unsuitable in cases of simple gastric irritation.

2. GASTRIC INSUFFICIENCY.

The term "atonic dyspepsia," although it includes most, does not include all the cases of gastric insufficiency.

Gastric insufficiency may be defined as a condition in which the functions of the stomach are diminished. The chief functions which are affected are the secretion of gastric juice and the motor activity of the organ, so that there is not only a greatly diminished chemical action on the food, but there is a greater delay of food in the organ than normal in the case of ordinary meals, owing to the diminished motor activity. It has been frequently explained in the previous chapters that gastric irritation may lead to gastric insufficiency, the overworked or the irritated organ at last showing a deficiency of function. In cases classed as gastric insufficiency, the condition has been induced, not by gastric irritation, but either by the mode of life or the age of the individual, or by the presence or antecedence of some diseased condition other than of the stomach.

Etiology.—Gastric insufficiency is developed in consequence of some general condition or disease of the body in which the stomach shares in the almost universal diminution of activity of the organs and tissues. Although the symptoms are directly brought out by the presence of food in the organ, yet in the cases under consideration, food and food accessories are not the cause of the gastric condition. It is induced in some by the character of occupation, by the mode of life, or by prolonged mental disturbance. Thus it is not uncommon in those who lead a sedentary life, with much mental work, especially if this be combined with business or social worry. Indeed, worry and the rush of modern life are responsible for a large number of cases of indigestion of food due to gastric insufficiency. These conditions, especially when associated with depressing surroundings and insufficient exercise, lead to a general depression of the functions, not only of the nervous system, but also of the other bodily functions. The appetite diminishes, the bowels become constipated, and the stomach soon shows signs of deficient activity. These causes act chiefly towards the middle period of life, and both in men and women, but more especially in men on account of the greater amount of mental and responsible work done. Indeed, from middle age and onwards during the period of old age, the functions of the gastro-intestinal tract are in the majority of instances less vigorous than in youth; it is the time of life when the daily amount of food required diminishes, and gastric insufficiency developing more or less suddenly, may show itself at this period for the first time. In these cases, excess of food or unsuitable food may be the actual agent in aggravating the insufficiency and bringing it to the notice of the patient. In women between forty-five and fifty years of age, at the onset of the climacteric, gastric insufficiency is frequently observed, and lasts in many instances until the disturbances of the menopause (uterine, nervous, and cardiac) are past, it may be during a period of two or three years. In prolonged lactation it frequently arises. In young adults, whether male or female, gastric insufficiency is commonly due to some recognisable morbid condition of the body. This functional disorder may be manifested early in life when inherited; for, although a difficult matter to correctly estimate, there is some evidence that a "weak" digestion may be transmitted from parents to offspring.

The general conditions of the body which may with other etiological factors induce gastric insufficiency are divisible into two classes: (1) non-febrile debilitating conditions or diseases, and (2) febrile conditions and diseases.

1. *Non-febrile Conditions.*—Gastric insufficiency is frequently noticed in cases where there are repeated losses of blood over a long period. Such hæmorrhage occurs from hæmorrhoids from which there may be frequent losses of blood for a long time; also in cases of profuse menorrhagia and metrorrhagia, commonly induced by fibroid tumours of the uterus. Repeated hæmorrhages from malignant growths, whether intestinal or external, also tend to induce gastric insufficiency. Prolonged suppuration, whether tubercular or syphilitic, as, for example, from bone disease, is a not uncommon cause. In hæmorrhage from a non-malignant disease, the condition of anæmia produced is no doubt responsible for the deficient

activity of the stomach. In prolonged suppuration, anæmia is present, but the primary disease, whether tubercle or syphilis, is also an etiological factor in the production of gastric insufficiency.

In anæmic diseases generally, whether primary, as chlorosis and pernicious anæmia, or secondary, as in chronic Bright's disease, malignant disease, and chronic pulmonary tuberculosis, the stomach functions frequently become deficient. In chlorosis, gastric insufficiency is well marked if the anæmia is pronounced; in pernicious anæmia, too, some cases show great deficiency in function of the stomach, bringing them into relation with cases of advanced atrophy of the organ. The indigestion of chronic Bright's disease is constantly only shown by gastric insufficiency; as in cancer and in chronic pulmonary tuberculosis. In gout, gastric irritation is more common than insufficiency, and when this develops it is usually to be attributed to a long period of gastric irritation, or to the effect on the body of renal disease.

2. *Febrile Conditions and Diseases.*—The state of fever tends to produce diminished functional activity of the stomach. This was noticed by Beaumont in the case of Alexis St. Martin, from the observation that in the febrile state the food remained twenty-four hours, or longer, in the stomach, and was undigested, while the mucous membrane showed a dry, glazed appearance. This diminution of functional activity lasts during the whole course of the fever, and is accompanied by loss of appetite. During convalescence a return of the functional activity of the organ is shown by an increase of appetite, but in this case especially the appetite is no criterion of the amount of food the patient can digest. The return of function to the normal is very slow, and is delayed frequently by injudicious feeding, and in some cases by continuing an insufficient diet for too long a period. During fever there is a deficient secretion of gastric juice, both of the pepsin and the hydrochloric acid, and a deficient motor activity. During convalescence, when, with an increase of the food, symptoms of gastric insufficiency are liable to develop, the effect on the stomach is not infrequently shown by a dilatation of the organ of moderate degree, and this dilatation, if un-

treated, may last a considerable time. This tendency to dilatation is a feature of these cases.

After most febrile diseases, gastric insufficiency may be observed, and last a variable time. After typhoid fever, it is perhaps most common, and after scarlet fever; but it is also observed after rheumatic fever and measles, and in the course of malaria. Gastric insufficiency occurring in chronic pulmonary tuberculosis is partly to be ascribed to the chronic febrile condition present, but also to the anæmia produced.

In most of the conditions above considered, it is evident that the gastric insufficiency is of temporary duration, being induced by some disease which has already passed away, or which is remediable by treatment. Some of the conditions, *e.g.* malignant disease, are incurable. But gastric insufficiency is sometimes associated with atrophy of the glandular and other structures of the stomach to a greater or less extent, so that it becomes a very grave disease (see Atrophy, p. 280). The majority of these cases occur beyond middle age, and are as a rule unrecognised during life. There are thus two classes of cases which may be called gastric insufficiency. In one, the condition is *temporary*, the original cause being remediable by treatment; in the other, the condition is *permanent*, being due to organic changes in the walls of the stomach, as in some cases of catarrh and in cases of atrophy and cancer of the stomach. It is not uncommon that in individual cases of primary gastric insufficiency, gastric irritation and gastric catarrh are induced by the action of the food as an irritant. Indeed, in such cases gastric irritation or catarrh is the natural sequence of the repetition of meals which the stomach cannot digest or manipulate.

Symptoms.—In gastric insufficiency, in response to the stimulus of food there is deficient secretion of the gastric juice (both acid and pepsin), and a deficient motor activity; and although both these functions may be fairly active up to a certain period of the digestion, they tend to cease before digestion is complete. There is therefore great delay of food in the organ. The repeated effect of unsuitable meals increases the deficiency of the organ, especially as regards its motor activity; dilatation therefore results. Lastly, the remains of the food in the organ are apt to undergo bacterial decomposition, especially if the organ is dilated (Chapters III. and XIV.). The symptoms referable to the stomach are chiefly associated with delay of food in the organ. After an ordinary meal there is a sense of weight and fulness in the epigastrium, such as has been described as occurring after gastric irritation. In gastric insufficiency, however, it is the mere presence of the food in the organ which causes the distress, so that these symptoms appear almost directly after a meal, and are not usually delayed for one or two hours, as in cases of gastric irritation. With the sense of epigastric fulness there may be an oppression of the chest, which sometimes gives rise to actual dyspnœa, especially on exertion. These symptoms as a rule last the whole time the food is in the stomach, commencing after a meal and lasting for two or three hours, or until the next meal, which gives no relief to the symptoms, as in cases of gastric irritation due to hyperacidity, but actually aggravates them. Flatulence is a constant symptom, and in the majority of cases it is not the flatulence of bacterial decomposition, but is due to an increased formation of the gases produced during normal digestion, or is due to regurgitation from the small intestine (p. 161). On the other hand, the flatulence may be due to bacterial decomposition of the food, chiefly, however, when the stomach is dilated.

Acid risings are as a rule absent. Towards the end of the period of digestion they may occur in gastric insufficiency, but they are not of that burning character they possess in gastric irritation. When acid eructations occur in the course of gastric insufficiency they are due to the onset of gastric irritation, induced by the constant presence in the organ of undigested or slowly digesting food, or they are due to an irritability of the organ induced by the general condition of the body. Vomiting is not a symptom of gastric insufficiency. Such patients may vomit, but this is as a rule due to some definite and ascertainable cause, such as the partaking of a particularly indigestible meal.

The reflex symptoms attributable to the indigestion of food in cases of gastric insufficiency are very numerous. It is in many cases difficult to ascribe their origin to the gastric condition, inasmuch as the general conditions of the body associated with gastric insufficiency, also play a part in their production. The reflex symptoms are chiefly referable to an effect on the nervous system (Chapter VII.). The diseases and conditions, however, discussed under the heading of etiology also produce nervous symptoms, and gastric reflexes are aggravated by the pathological condition present in these diseases. Thus the condition of the nervous system induced by overwork, worry etc., by chlorosis and anæmia generally, and in the convalescent stage of acute diseases itself, aggravates and exaggerates the reflex symptoms of gastric insufficiency. Moreover, these gastric reflexes are associated with symptoms produced by the nervous condition. It is to be noted, however, that the reflex effects ascribable to gastric insufficiency are related in point of time with the slow process of digestion in the stomach. This is especially so with the pain in the front of the chest, the pain between the shoulders, and that "below the heart," which is frequently directly associated with flatulence, or the presence of a large amount of gas in the stomach. Pain may be felt in other situations in various parts of the chest, chiefly in the lower axilla (commonly in chlorosis); in the head, due to constipation, or to a general condition of the nervous system; in the abdomen, due mainly to constipation. Drowsiness after meals is a common symptom (Chapter VII.), also sleeplessness (*loc. cit.*), both of which have been already fully discussed. A feeling of lassitude, disinclination for exertion, is frequent, as well as mental inaptitude, apathy, and a melancholy view of life. These mental effects are, however, due chiefly to the general condition or disease producing the functional disorder and not to the gastric insufficiency itself. This aggravates the symptoms but does not produce them.

The *face* frequently shows the general condition associated with gastric insufficiency; it is often pale, perhaps anxious-looking, but not expressive of pain. The complexion is frequently "muddy." In chlorosis and other forms of anæmia, and in chronic Bright's disease, the face shows the characteristics of the particular disease; in chlorosis, the greenish-yellow tint; in chronic Bright's disease, the opaque pallor of the face, with baggy lower eyelids. The *appetite* is always diminished.

The *tongue* is commonly broad, pale, flabby, and tooth-indented, the tongue not only of anæmia but of anæmia-producing diseases or of individuals simply "run down." It may be coated with a light fur, but is not infrequently clean. Thick coating of the tongue is not a sign of gastric insufficiency, and when present may be due either to gastric irritation or catarrh, chronic tonsillitis and pharyngitis, or to fever. Enlargement of the tonsils is not infrequently present in cases of gastric insufficiency, especially when occurring in children near puberty, who show slight signs of gastric disturbance and constipation, with pallor and lassitude.

The *bowels* are usually constipated. Constipation is the rule in gastric insufficiency, and is no doubt for the most part due to deficient motor activity of the intestines. The bowels are opened once in two or three days, or only once a week in women, or even once a fortnight. As in the case of gastric irritation, constipation may alternate with looseness of the bowels. But in this case, the looseness does not mean that the colon is emptied, but it is due to the irritation of the scybala in the gut; so that such cases are best remedied by aperients. Diarrhœa may be present in gastric insufficiency, when due to a special cause, as in chronic pulmonary tuberculosis and in the convalescence of typhoid fever; it is, however, not a symptom of the stomach disorder.

The *pulse* is weak, it is not very frequent, although the frequency may be readily increased on exertion or on any sudden emotion. It is regular and compressible. The diminution of arterial tension is due to the general condition, and may be quite unassociated with any organic cardiac disease. On the other hand, the pulse may be characteristic of the general disease, as the high-tensioned pulse of chronic Bright's disease.

Effect on the General Nutrition.—It is difficult in most cases to gauge the effect of gastric insufficiency on the general nutrition of the body, inasmuch as the stomach condition is only a part, and frequently a small part, of a disorder or disease of another part of the body. If the stomach disorder is well marked, owing to the insufficient quantity of food which the patient assimilates, wasting occurs; and even when not well marked there can be little doubt that gastric insufficiency increases the loss of weight which is produced by the general disease. This is not uncommonly seen in cases of chronic pulmonary tuberculosis in which the disease is progressive and the emaciation is progressing, while there are the symptoms of inability to digest the food. By dietetic and by medicinal treatment the stomach condition improves, and frequently an observable change occurs in the general nutrition. Chlorosis, again, is a disease which, when existing by itself, does not lead to wasting, owing, as in other forms of anæmia, to the diminished amount of oxygen carried to the tissues; yet if there is well-marked indigestion of food, either gastric irritation or gastric insufficiency, some wasting may be observed. Here, again, as in chronic pulmonary tuberculosis, a great improvement is observed by relieving the gastric symptoms and thus enabling more food to be digested. In gastric insufficiency induced by the mode of life, there is some degree of wasting due as much to the condition of the nervous system as to that of the stomach. Some of the most intractable cases are, however, those that arise during the convalescent stage of acute febrile diseases, or in the course of a chronic febrile disease, such as chronic pulmonary tuberculosis. Thus after typhoid fever the stomach disorder may last a long period—months—during which time the patient does not regain his weight and remains in a weak condition. After a slight attack of malaria or of "low fever," gastric insufficiency may be very intractable, owing chiefly to irregularities in diet and want of care in regulating the mode of life. It may be said generally that temporary gastric insufficiency does not commonly cause any great degree of emaciation, although it is an aid to the primary disease in producing loss of weight.

Permanent gastric insufficiency is, however, associated with great emaciation. These are cases where there is degeneration of the glands of the stomach (p. 214), and are discussed under the headings of gastritis (Chapter VIII.), of atrophy (Chapter IX. p. 280), and of cancer (Chapter XVI).

Course and Prognosis of the Disorder.—The gastric insufficiency of old age and of those who have inherited a weak digestion is a more or less permanent condition, the effects of which, however, are held in check by the use of a proper diet

—a diet regulated to the amount of work the stomach can perform, and also by medicinal and hygienic treatment. In all cases of gastric insufficiency the course and prognosis of the stomach condition depend on the amenability to treatment of the general condition or disease producing the stomach disorder. This may be kept in check as regards the production of symptoms, but any permanent improvement is not to be expected until an amelioration of the general disease occurs. Thus in chronic pulmonary tuberculosis, as long as there is fever, with signs of the spreading of the lung disease, so long may the gastric insufficiency be present; an abeyance of the fever, a tendency to quiescence of the lung disease, leads to a better digestion and a better prognosis. One of the chief conditions which influences the prognosis of gastric insufficiency, as indeed it influences all curable gastric affections, is that of neglect. Carelessness in the mode of life, in the regulation of meals, and in the character of the food and food accessories taken lead in time to permanent insufficiency, a condition not readily amenable to relief by treatment. The keeping of these conditions indeed depend more on the patient than on the practitioner, and their breach is especially observable in cases where there is a great "hurry of life" and in the convalescence from acute diseases.

Treatment.—Medicinal treatment is directed to the improvement of the stomach conditions by stimulating secretion (Chapter X. p. 290), by increasing the movements (p. 293), by counteracting bacterial fermentation, and by treating dilatation (Chapter XIV.). Dietetic treatment is fully discussed later (Chapter XI. p. 331). In temporary gastric insufficiency treatment is very effectual; in permanent gastric insufficiency it is only palliative. In both the results of treatment depend largely on the removal or the curability of the primary condition.

CHAPTER VIII.

MECHANICAL AND ACTIVE CONGESTION OF THE STOMACH—GASTRITIS—GASTRIC CATARRH.

In the previous chapters, indigestion of food has been treated as a purely functional disease, due either to the food taken and the abnormal changes it undergoes, or to variations in the functions of the organ (secretion and motor activity) from the physiological standard of the individual. Indications have been given to show that these functional changes may be associated with anatomical changes both in the glandular and muscular structures of the organ; and a closer study of these changes is now necessary.

The secretion of the gastric juice depends, as has been shown, on a very delicate mechanism. It may be repeated that in this process the pyloric portion of the stomach differs from the cardiac, inasmuch as, although it secretes pepsin, it does not secrete hydrochloric acid, the secretion of both substances being the function of the cardiac region. A continuance of the secretion during nearly the whole period of digestion is of prime importance in normal digestion, and depends not solely on the anatomical integrity of the secretory glands, but on a continuous blood supply and on the nervous mechanism. It is quite clear from this how important slight anatomical changes in the glands may be in disease, especially when associated with any variation in the amount of blood supply during the time the food is in the stomach, and any changes in innervation. Something has already been said regarding the irresponsiveness of the stomach to stimulus in disease. In cases where the stomach does not respond to stimulus, not only the secretion of gastric juice, but the blood supply and the mechanical movements of the stomach are deficient. Conditions of blood supply probably play a very great part in the production of the symptoms of disordered digestion, any alteration, whether in the form of anæmia or congestion, affecting all the functions, the secretory, the motor, and the absorptive. Anæmia probably produces only functional disturbances, but both mechanical and active congestion (inflammation) lead to anatomical changes, which have a more profound effect on the functions of the organ.

The stomach is peculiarly situated as an organ, inasmuch as, being the receptacle of all the food received into the body, it is exposed to various kinds of irritants, some of which are slight, some great, and many of which produce their effects only after long-continued indulgence (*e.g.* alcohol) and long-continued abuse of dietetic rules (see Food as an Irritant, Chapter III. p. 68).

In the second place, when irritant or corrosive poisons —such as the mineral acids, the caustic alkalies, oxalic acid, carbolic acid, phosphorus, arsenious acid, corrosive sublimate, and a few others—have been taken into the stomach, the original effects, those of acute inflammation of the organ, not infrequently pass off leaving a severe chronic condition. In certain cases, also, an acute affection of the organ results from the invasion of its walls by bacteria either through the mucous membrane or from a septic focus already present in the body.

Lastly, acute and chronic inflammation of the stomach are present in certain acute diseases, in which there is no direct invasion of the stomach walls by infective micro-organisms, but in which there is an inflammation of the stomach resulting in all probability from the circulation in the body of the chemical poisons produced in the infective disease.

These conditions may be grouped under the term gastritis as follows:—

Acute gastritis.	1. *Simple,* occurring in certain infective disorders, or due to disorders of diet. 2. *Toxic,* gastritis toxica, due to poisons. 3. *Infective,* gastritis mycotica, due to bacterial infection.
Chronic gastritis or catarrh.	Occurring as a sequel of the acute stage, or chronic from the first.

The gastritis occurring as the result of the invasion of bacteria is a special variety which will be treated separately, under the heading of Bacterial Infection of the Stomach, Gastritis Mycotica (Chapter IX.). Toxic gastritis is also a special condition differing from that occurring as the result of irritant food or in infective disorders (Chapter IX.). The gastritis of the last two varieties is best termed gastric catarrh, acute and chronic.

ETIOLOGY.

In many individual cases of disease, the various pathological factors which end in producing catarrh of the stomach cannot be determined with accuracy, but in nearly all cases the principal etiological condition ending in catarrh is usually evident.

As a pathological factor in the production of catarrh, irritation stands first. Such irritation is shown in its most advanced form in irritant poisoning by the substances previously mentioned; in its milder forms, the irritation is produced by a variety of causes. In these last forms food has been shown to act as an irritant, not only from its bulk, but from its chemical composition and from its being mixed with an excess of food accessories. The abuse of food accessories is responsible for a large number of cases of catarrh of the stomach, both acute and chronic. Teetotallers are, however, by no means exempt from the disease. Food is not an irritant which in most instances acts in a short time, but the prolonged breach of dietetic rules leads to irritation of the organ.

In the second place, an increased acidity of the gastric contents acts as an irritant to the mucous membrane. This increased acidity is of three kinds, as has been shown (Chapter

IV. p. 81); (1) due to an excessive secretion of hydrochloric acid, (2) to an excessive amount of organic salts and acids taken with the food, or (3) to an excessive formation of organic acids (lactic, butyric, and acetic) by the bacterial fermentation of carbohydrates in the stomach.

Lastly, the long duration of food in the stomach, *i.e.* the delay of food after digestion is completed, acts as an irritant to the mucous membrane, especially when the stomach contents remain excessively acid as they do in bacterial fermentation.

These factors, often in combination, play a great part in producing irritation of the mucous membrane, and their effect is aggravated when disordered digestion is already established, by the presence of dilatation of the stomach and by an excessive formation of mucus by the mucous membrane.

The first effect of mild irritation on the gastric mucous membrane is twofold; it increases the amount of mucus secreted, and it increases the secretion of gastric juice. This is a condition of things as regards the secretion of gastric juice, which has already been discussed (Chapter IV., Hyperacidity due to HCl). The irritation in this case, caused by food, may be simply regarded as an increased stimulation of the mucous membrane, leading to increased secretion.

A long continued mild irritation or a greater irritation produces, however, definite anatomical changes in the mucous membrane—a catarrhal inflammation, with changes in its functions, chiefly in the way of diminishing them.

A close examination of the etiology of gastric catarrh shows that, although food and food accessories are always the direct exciting causes of the inflammation, yet it is predisposed to by a large number of diseases and conditions both outside the stomach and in the organ itself. Acute catarrh, a not very common disease, arises suddenly when an excessive irritant is taken into the stomach in a general disease which greatly predisposes to it, or it may arise as an acute exacerbation of chronic catarrh. Chronic catarrh may succeed the acute form or be chronic from the first. The etiology of both forms will be considered together under the following headings:—

1. As a sequence of functional disturbance of greater or longer duration. It is chiefly in gastric irritation that catarrh follows functional disorder, and for reasons many times explained in the previous chapters. In gastric irritation in its severe forms, the irritation is not only that of the food and food accessories, but that of hyperacidity of the stomach contents due to hydrochloric acid, and of a long-standing hyperacidity. This condition frequently leads to catarrh, chiefly chronic, and may end in a permanent injury to the organ, due to inflammatory changes in the mucous membrane. Acute catarrh may also arise in this way, but there is usually a special reason for its incidence, *e.g.* some particular indiscretion in diet. Catarrh may follow gastric insufficiency. It was seen that gastric irritation may be added to gastric insufficiency owing to dietetic irregularities; catarrh may also follow in this way. In gastric insufficiency, however, the production of chronic catarrh is not associated with hyperacidity due to hydrochloric acid, but is largely dependent on the delay of food in the stomach, and, when dilatation is present, with bacterial fermentation of the food, which ends in the formation of irritating organic acids (see Dilatation of the Stomach, Chapter XIV.). Catarrh does not so frequently follow gastric insufficiency as it does gastric irritation. In both cases its development is an important feature, since it may lead to serious anatomical changes in the organ.

2. Inflammation, whether acute or chronic, does not depend altogether on the strength of the irritant; it may be predisposed to by a condition of the organ, brought about by a local or general disease. Thus acute gastric catarrh may be observed in tuberculosis, scarlet fever, measles, rickets, pyæmia, and septic diseases (puerperal fever, etc.); in pneumonia, typhoid fever, malaria, and in cholera. It is, however, in the convalescent stage of acute diseases that catarrh of the stomach is likely to arise, and usually in a chronic form. Patients, indeed, often date their stomach trouble from some acute illness they have had. In the convalescent stage, as has been shown, the stomach is recovering from the state of functional insufficiency which is present during the acute febrile stage of the illness. Irregularities of diet in the convalescent stage,

while they may produce only the symptoms of gastric insufficiency, may also lead to gastric irritation and gastric catarrh. Besides this, there appears to be a predisposition to inflammation of the stomach in some of the diseases named, although anything very definite cannot be said on this point. In 100 stomachs examined by Wilson Fox,[1] acute or chronic catarrh was found in 57. Thirty-three of these occurred in febrile or septic diseases, 12 of which were tuberculosis of the lungs, the others being pneumonia, septic diseases, abscess, cholera, and typhoid fever. The other 24 cases were chronic and non-febrile diseases; cardiac disease, with or without bronchitis, chronic Bright's disease, hernia, diabetes, cirrhosis of the liver, delirium tremens, alcoholism. These results show how frequent catarrh of the stomach is; but it cannot be said that they necessarily show any special connection between the particular diseases and the inflammatory condition of the stomach. Some of the diseases, inasmuch as they produce anæmia and a general state of malnutrition, may predispose to gastric catarrh, viz. chronic Bright's disease and chronic pulmonary tuberculosis. Cardiac disease and cirrhosis of the liver may more directly predispose to catarrh, since they lead to mechanical congestion of the organ (see p. 229). It may be said, therefore, that while arising in some chronic diseases, catarrh is frequently associated with acute febrile diseases, arising during the acute stage or more frequently during the stage of convalescence. As regards gout, the gastric irritation so often present in this disease frequently proceeds to catarrh, and the stomach condition is aggravated by the general condition of the body, perhaps by the excess of uric acid.

Catarrh is a frequent accompaniment of other organic diseases of the stomach. The injury of the mucous membrane produced by irritant poisons leads to a prolonged inflammation of the organ in cases of recovery from the poison (Chapter IX.). In chronic ulcer of the stomach, and more especially in carcinoma, catarrh may arise. In both conditions there is a mechanical injury to the organ produced by disease, hampering its movements, altering its natural secretion. This local injury no doubt leads to variations in the normal blood supply in response to the stimulus of food, and to local inflammatory changes around the injured spot, whether a simple ulcer or a carcinomatous. Irritation of such an injured organ is therefore likely to set up inflammation, which in the case of the stomach is prolonged since the irritation is continued. In ulcer of the stomach gastric irritation is a common symptom, and is seen in its most marked forms; the irritation is due partly to the presence of the sensitive ulcer, and partly to the hyperacidity of the stomach contents due to excessive secretion of hydrochloric acid (see Chapter III.; and Ulcer, Chapter XV.). Catarrh, however, is not frequently present in the early stages of ulcer, although in the late stages it may be well marked. In carcinoma ventriculi, besides the presence of the growth, there is an irritating discharge from the ulcerated surface, and in cases of pyloric obstruction a dilatation of the organ, in which bacterial fermentation of the food occurs, and in which the mucous membrane is exposed for long periods to the irritating action of an excess of organic acids. Catarrh, therefore, is a frequent accompaniment of carcinoma ventriculi.

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[1] Reynold's "System of Medicine," vol. ii. p. 890.

Lastly, irritation of the stomach leading to congestion and catarrh may ensue from pressure on the organ from without, and by surrounding inflammation. Examples of pressure occur in abdominal new growths near the organ, some of which press upon the organ near the pylorus; of surrounding inflammation, in general peritonitis and in chronic tubercular peritonitis.

It is evident from a consideration of its etiology, that catarrh of the stomach is a disease not closely related to the age, sex, or temperament of the individual. Both in its acute and chronic forms it may be met with in children and in adults, in men and in women. Climate and race have no influence in the production of the disease, except in so far as the mode of life and the diet are such as to produce inflammation of the stomach.

PATHOLOGICAL ANATOMY.

The examination of the stomach presents great difficulties after death. A post-mortem examination being made usually about twenty-four hours after death, the stomach very commonly presents signs of auto-digestion and of post-mortem staining, conditions which render a physiological or microscopical examination quite useless. In some instances, however, these post-mortem changes do not occur, especially when there is an atrophic condition of the glands of the mucous membrane, so that no gastric juice is secreted. As a rule, however, unless the examination is made a few hours after death, or unless shortly after death the stomach is washed out and filled with weak alcohol, both the naked eye and the microscopical examinations, especially the latter, are of but little value. Indeed, many of the changes in the mucous membrane of the stomach which were formerly ascribed to disease were in reality post-mortem changes and auto-digestion. The diagnosis of congestion of the stomach after death is thus extremely difficult in many cases. Sometimes there is no doubt of the condition, as when the mucous membrane is of a pink colour, and the contents of the organ consists of tenacious mucus, and little else. This active congestion of the organ is not commonly seen after death, and has been observed in infective disorders. It is one of the phases of some acute gastric attacks, and as these are for the most part curable, it is not commonly observed post-mortem. Even when there has been extreme mechanical congestion of the stomach during life, such as results from obstruction to the flow of blood in the portal system, the stomach frequently does not show any well-marked signs of congestion after death. In both mechanical and active congestion of the organ the results of the condition must therefore be looked for. It may be, as occurs in some cases, that well-marked mechanical congestion has existed during life, as shown by hæmatemesis, and that after death no naked eye, or even microscopical sign is seen of the condition. This is one of the difficulties that has always existed in the study of the pathology of diseases of the stomach; pathological conditions not observed post-mortem had to be deduced from the symptoms the patient experienced during life, with the result of the development of many erroneous ideas regarding the organ.

Post-mortem Softening of the Stomach (*Auto-digestion, Gastro-malacia*).—This occurs when at death the stomach contains food in the process of digestion, and is due chiefly to the action of the gastric juice on the dead or dying tissue. It occurs at varying intervals after death and is accelerated by a high external temperature, and to some extent by the cause of death, since in cerebral disease (tumour and meningitis) it appears to begin even before the final evidences of death are manifest.

The appearances met with are very characteristic. They consist chiefly of softening of the walls of the organ with transudation of the hæmoglobin in the blood vessels and its transformation into hæmatin. The mucous membrane, especially at the cardiac end, is softened and destroyed over a greater or less area, and the whole of it may be lost. Sometimes the loss of substance occurs in small areas not larger than two or three millimeters in diameter, as if the auto-digestion had occurred chiefly in the small depressions into which the gastric glands open. These areas are seen only at the cardiac end of the organ. The softening process may extend through all the coats, causing a rugged rupture of the organ and an extrusion of its contents into the peritoneal cavity, and the part of the diaphragm opposite the rupture may also be softened and digested. The discoloration of the mucous membrane due to the solution of the hæmoglobin and its transudation presents very characteristic appearances. Along the rugæ, and especially at the cardiac end, there are dark-red or black-red lines which show the parts where the hæmoglobin has been most affected by the acid contents of the organ. Frequently, too, the vessels are mapped out by a surrounding dark-red area, which is very characteristic of post-mortem softening.

In the latter stages putrefactive decomposition is added to the changes induced by the gastric juice.

Mechanical Congestion of the Stomach and its Results.

Mechanical congestion of the stomach arises as the result of portal obstruction; more commonly directly from pressure upon the portal vein itself, from thrombosis of it, or from cirrhosis of the liver, less commonly as the result of dilatation of the right side of the heart from whatever cause, or of pressure on the vena cava inferior near its entrance into the right auricle.

From the arrangement of the veins of the organ it is clear that obstruction to the flow of venous blood would affect the pyloric part of the stomach in the first instance, because the veins from this part flow directly to the portal system, while the venous capillaries of the cardiac region are connected with those of the lower end of the œsophagus, and in this way the congestion of the cardiac region is in part relieved. In long continued mechanical congestion the effect on the pyloric region is seen in pigmentation, which may be quite absent from the cardiac region. The effect of mechanical congestion will be best illustrated by the following example which occurred in a boy, a teetotaller, who died from advanced atrophic (multilobular) cirrhosis of the liver. During life there was

FIG. 18.—Pitting of the mucous membrane in mechanical congestion. From a photograph, about natural size. The preparation was from a case of advanced cirrhosis of the liver in a boy.

hæmatemesis, and shortly before death rapidly advancing ascites. The liver presented the usual features of cirrhosis, the disease being very advanced; the spleen was enlarged, and showed the results of mechanical congestion. The stomach was moderately dilated, and contained a thick, dark, grumous liquid, the remains of food mixed with altered blood. One of the features of the naked-eye appearance of the mucous membrane was pigmentation of the whole of the pyloric region, occurring in dots over small areas from one to two lines in diameter, and regularly distributed. No areas of pigmentation were observed in the cardiac region. The pyloric region was otherwise pale, but the cardiac region showed areas of patchy congestion of a dull red colour. A second feature of the naked-eye appearances was the presence chiefly in the mucous membrane of the cardia, but also in that near the pylorus (the middle region of the organ being more or less free) of numerous small pits or erosions of the membrane, not so deep as the submucous tissue (Fig. 18). In many of these pits, which were irregular in shape, and varied between one and two lines in diameter a small, red blood clot was visible, showing the origin of the hæmorrhage during life. Microscopically, these erosions were seen to be pits in the mucous membrane, due to a super-

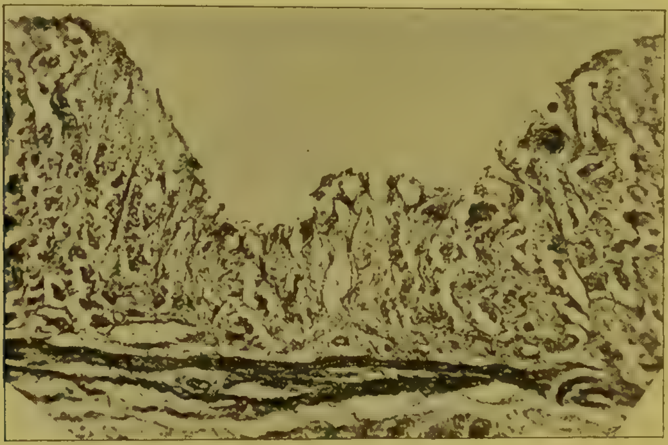

FIG. 19.—Microscopical appearances of the specimen represented in Fig. 18. From a photograph, × 75. The figure shows that the erosions were quite superficial, being limited to the mucosa. The appearances are very similar to those occurring in auto-digestion of the mucous membrane of the stomach. From a preparation hardened in Müller's fluid, and stained with logwood.

ficial necrosis of the glands (Fig. 19). Four inches from the pylorus on the posterior aspect of the organ was an oblong, smooth scar in the mucous membrane, $\frac{1}{4} \times \frac{3}{4}$ inch, quite superficial (limited to the mucous membrane), with no puckering around. It was the scar of an erosion of the mucous membrane.

The effects of chronic congestion were also seen in the small intestine, the mucous membrane of which was pigmented throughout.

The chief features therefore of mechanical congestion of the stomach are:—

1. A congestion of the mucous membrane, especially of the pyloric part, with submucous ecchymoses, which become pigmented areas.
2. Minute erosions of the mucous membrane, from which bleeding may occur; larger erosions, which heal with a smooth scar without puckering. The ordinary ulcer of the stomach is not seen as a result of mechanical congestion.
3. Dilatation of the organ, owing to weakness of the middle coat.

Milder degrees of the affection are associated only with a diminished activity of the gastric glands and with slight dilatation of the organ, and this may be considered the condition in those cases of cardiac disease with dilated right heart and in most of the cases of cirrhosis of the liver in which gastric symptoms are the consequence of mechanical congestion of the organ.

Active Congestion and Catarrh with their Consequences.

1. *Acute Catarrh* (*Gastritis Catarrhalis*).—When a mucous membrane becomes acutely inflamed, it is swollen and congested sometimes uniformly or more frequently in patches, and although in the very early stage it is dry, its surface is soon covered by an exudation more or less tenacious. When a microscopical examination is made of a mucous membrane in this condition, the vessels in the submucous tissue are seen greatly

distended, and surrounding them and infiltrating the tissues around are numerous leucocytes which have escaped from the blood vessels. The interstitial fluid is also increased. The leucocytes are increased up to the basement membrane on which the epithelial cells rest. These changes are the results of the first stage of inflammation, in which there is congestion, exudation of lymph, and migration of leucocytes. The effect of these changes are shown on the epithelial cells forming a covering for the mucous membrane, and on the cells lining the glands situated in the submucous tissue. The epithelial cells lining the membrane show an increase of the goblet cells which produce mucus and are normally present in small numbers. The increased secretion of mucus accumulates on the surface as round droplets or strings until it is removed (Fig. 20). This mucous secretion contains cells which at first are the epithelial cells with some leucocytes. As the process goes

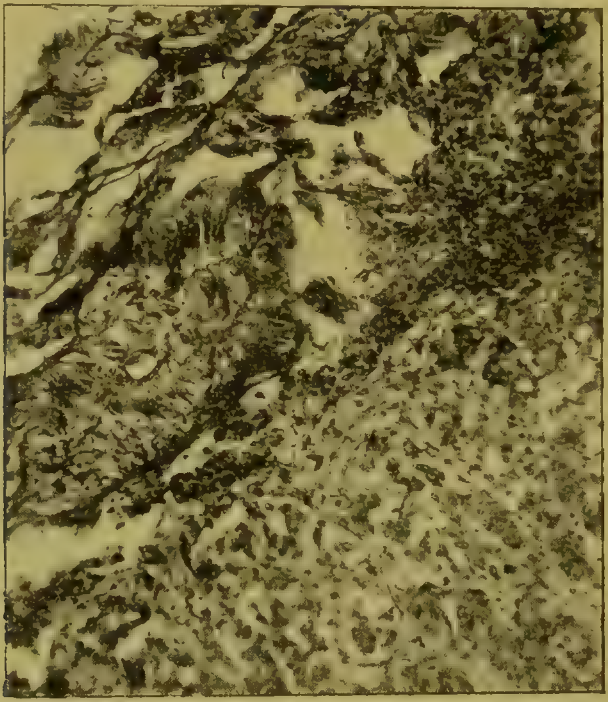

Fig. 20.—Early stage of catarrh of the stomach, in a child who died from rickets, cranio-tabes, and tetany. From a photograph, × 125. The mucous membrane is seen to be covered by strands of mucus, which enclose the epithelial cells: many of these are goblet cells. On the right of the figure and above, there is a large collection of leucocytes, partly near the surface of the mucous membrane and partly in the mucus covering this. From a preparation hardened in osmic acid and stained with logwood.

on the number of leucocytes (pus-cells) in the mucus increases. The cells lining the glands situated in the submucous tissues also undergo a varied transformation; they swell up so as in many cases to fill the lumen of the gland, their nuclei become indistinct, and in them are seen numerous droplets which coalesce and are finally extruded from the cell as mucus. The extent to which the glands are affected varies with the degrees of inflammation, and normal glands may be seen in the same specimen side by side with those extensively changed in the manner described (Fig. 21).

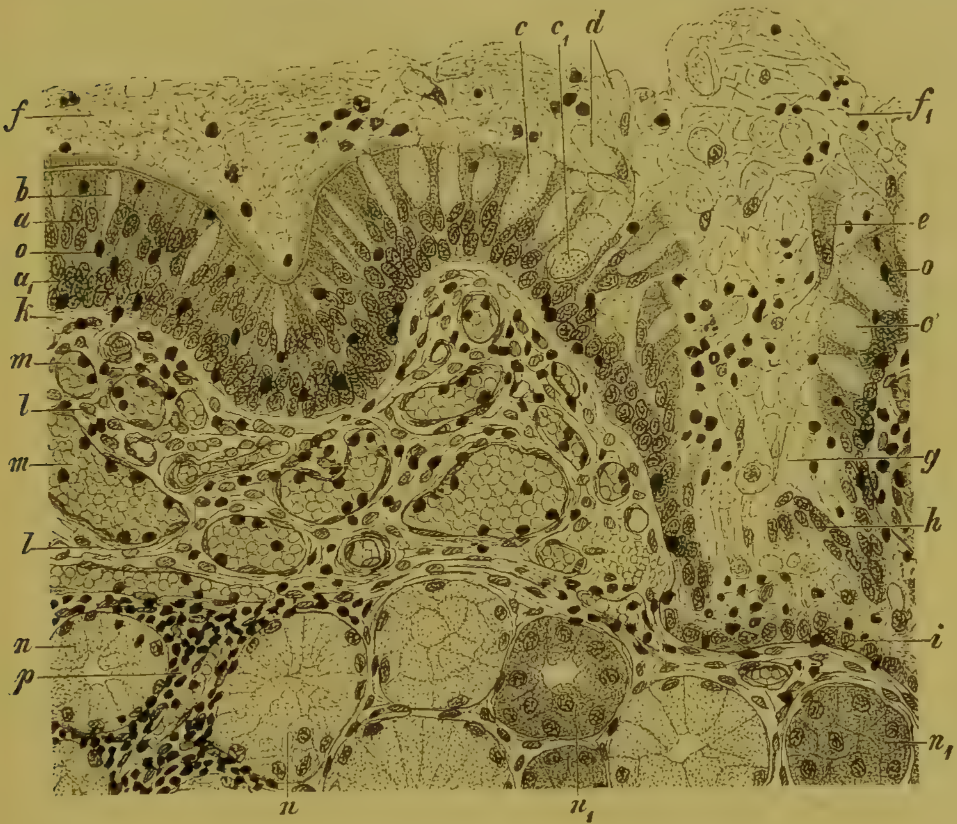

FIG. 21.—Superficial catarrhal inflammation of a bronchus: × 120. *a*, ciliated epithelial cells; a_1, deeper cell-layer; *b*, goblet-cells; *c*, c_1, cells showing advanced mucoid formation; *d*, mucous cells with mucoid nucleus; *d*, mucoid cells, cast off from mucous membrane; *e*, cast-off ciliated cells; *f*, and f_1, mucous exudation on surface containing pus corpuscles; *g*, opening of a gland full of mucus; *h*, shed epithelium of gland; *i*, epithelium still intact; *k*, swollen basement membrane; *l*, interstitial tissue of the mucous membrane; *m*, dilated blood vessels; *n*, glands full of mucus; n_1, glands without mucus; *o*, leucocytes in the epithelial layer; *p*, small-celled infiltration of the interstitial tissue of the glands. (Ziegler.)[1]

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[1] Ziegler, "Lehrbuch der Pathol. Anatomie," 5th ed., vol. i. Fig. 84.

As the process extends there is more and more mucoid degeneration of the epithelial cells on the surface, and eventually the cylindrical epithelial cells may be in greater part shed, leaving only the round cells at their base. When this has happened, the secretion of mucus diminishes, and there is an increased exudation of leucocytes, so that the secretion of the mucous membrane becomes more purulent.

In the process of recovery from this inflammation, the congestion diminishes, the leucocytes disappear, the epithelium of the membrane, as well as that of the glands, is regenerated, while the secretion becomes less and less purulent, and more mucoid, and diminishes until it is normal in amount.

During an acute inflammation of a mucous membrane, therefore, an acute catarrh, as it is called, the secretion is in the early stage watery and contains a large quantity of albumin, it then becomes mucoid and muco-purulent, and at the height of the process chiefly purulent, with a little mucus; during recovery the pus diminishes, and mucus takes its place.

All the changes in a catarrh are well seen in the nasal and the bronchial mucous membrane. In the latter case, there are in many cases only the signs of bronchial irritation with excessive secretion of mucus; in other cases there is definite catarrh, with very slight expectoration, occasionally muco-purulent; and finally acute and chronic bronchitis, the former going through the phases just described as characteristic of acute catarrhal inflammation. The great difference, however, between the effects of a catarrh of the bronchial tubes and those of the gastric mucous membrane is that the latter is composed chiefly of glands, whose secretion is a necessity for digestion, the absence of this secretion being a source of severe symptoms. Catarrh of mucous membrane increases the activity of the mucin-forming cells, but it diminishes the secreting activity of the cells of the gastric glands. This it does partly by the condition of congestion, for active congestion of a part means stasis of the circulation of the blood, and therefore, in such a case, there is not that constant and increased supply of blood to the mucous membrane which is a necessity for the continued secretion of the gastric juice. But there is another condition which is more serious; the glandular epithelium which normally produces the pepsin and the hydrochloric acid becomes affected by the inflammation, swelling up, and becoming granular or producing mucin. When they undergo the mucinoid or the granular degeneration, these cells do not produce pepsin and hydrochloric acid. In the considerable number of cases of acute and chronic catarrh of the stomach which have been microscopically examined, the mucin transformation of the cells has been sometimes found, but more commonly the granular degeneration. In this latter condition the cells become extremely granular and swollen, resembling in appearance the cells of the liver or kidney which have undergone cloudy swelling (p. 242). The granules are at first soluble in diluted acids and alkalies (Wilson Fox),[1] but subsequently fat globules only are seen, and the cells may completely degenerate. Both these changes in the secreting cells—the mucin degeneration and the granular degeneration—lead to their diminished activity, and are the cause of the great diminution or absence of pepsin and hydrochloric acid in the stomach contents which has been previously considered (Chapter IV.).

In acute catarrh of the stomach, erosions and superficial ulcerations of the mucous membrane not infrequently occur. These ulcerations occur through a loss of the superficial epithelium and of the layer of cells below, and of the superficial part of the glands. The ulcerations are never very deep, extending as far only as the mucous membrane and not down to the submucous tissue. These superficial ulcers are also formed through the solitary glands which exist in the mucous membrane, especially of the pyloric region. In acute catarrh these are enlarged and the cells may undergo degeneration, and an erosion of the superficial part of the gland occurring, the cells are lost, leaving a pit in the mucous membrane, constituting the ulcer.

The account given by Beaumont of the appearances of the mucous membrane of the stomach during an acute attack of gastritis is the only one describing the changes observed during life.

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[1] Art. "Diseases of the Stomach," in Reynold's "System of Medicine," 2nd ed., 1872, p. 879.

His words may be quoted in full[1]:—

"There are sometimes formed in the internal coat of the stomach eruptions or deep red pimples, not numerous, but distributed here and there upon the villous membrane, rising above the surface of the mucous coat. These are at first sharp-pointed and red, but frequently become filled with white purulent matter. At other times irregular circumscribed red patches, varying in size and extent from half an inch to an inch and a half in circumference are found on the internal coat. These appear to be the effect of congestion of the minute blood vessels of the stomach. There are also seen at times small aphthous crusts in connection with these red patches. Abrasion of the living membrane, like the rolling up of the mucous coat into small shreds or strings, leaving the papillæ bare for an indefinite space, is not an uncommon appearance. These diseased appearances, when very slight, do not always affect essentially the gastric apparatus; when considerable and particularly when there are corresponding symptoms of disease, as dryness of the mouth, thirst, accelerated pulse, etc., no gastric juice can be extracted." Beaumont describes the secretion from such a stomach in the following words:—"The gastric fluids extracted were mixed with a large proportion of thick ropy mucus, and considerable muco-purulent matter slightly tinged with blood, resembling the discharge from the bowels in some cases of chronic dysentery." There is no doubt from the description that the appearances were due to an acute catarrh of the stomach; they give a vivid picture which ought to be fixed in the mind of every practitioner who has to treat a case of acute gastric catarrh.

2. *Chronic Catarrh of the Stomach—Chronic Gastritis.*—A chronic catarrh is either a result of a definite acute attack, or it may be chronic from the first, although in this case it is probable that the early acute stages were slight and overlooked or consisted only in irritation of the stomach. The features of a chronic catarrh are chiefly two in number:

1. There is a continuous and excessive secretion of mucus.
2. There is more or less degeneration in one or more forms of the proper gland tissue of the mucous membrane.

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[1] *Op. cit.*

Therefore with the excessive secretion of mucus, there is a diminished secretion of gastric juice.

There are very varying degrees of chronic catarrh of the stomach. Those forms that have been seen post-mortem and are described as typical of the condition are the advanced cases. The milder cases, which are so common in practice are not seen post-mortem, and the exact degree of pathological changes present in them is chiefly a matter of inference.

There is first an increased secretion of mucus, not however from all parts of the mucous membrane, but from areas of varying extent. In chronic gastric catarrh one part of the mucous membrane is more affected than another. There are seen in some cases patches of congestion marking these areas; but frequently no congestion is seen when from the symptoms and the general pathological condition there was but little doubt of its existence. Pigmentation is sometimes seen, chiefly of the pyloric area, and in a form precisely similar to that occurring in mechanical congestion (p. 230). The pigmentation occurring in chronic catarrh has the same origin as that in mechanical congestion, and is due to small effusions of blood in the mucous membrane. In acute catarrh, as has been seen, besides the congestion of the mucous membrane, there is also a small-celled infiltration in the mucous and submucous tissue, and a degeneration of the epithelial lining of the stomach and of the cells of the secretory glands, this latter degeneration being in two forms, a mucin degeneration and a fatty degeneration. In chronic catarrh, these changes may progress to the formation of two different conditions, both of which have the same result, viz. destruction of the glands secreting the gastric juice. There may be an increase of the interstitial connective tissue, producing a condition of *fibrosis of the mucous membrane*, or the chief change may be a *parenchymatous degeneration of the gland tissue.* Not uncommonly both conditions are seen in the same specimen.

The naked-eye examination of the stomach in chronic catarrh, besides the signs of congestion and pigmentation already described, shows in some cases that the mucous membrane is tougher than natural, tearing with difficulty;

in others, that it is dead yellow in colour, soft and friable ; it is usually thickened, but is sometimes thinner than normal, and does not show any signs of auto-digestion. Frequently, however, no morbid change can be seen with the naked eye, the condition being only discovered by means of the microscope. The stomach may or may not be dilated, in some cases being smaller than normal (as in cases of advanced cirrhosis of the organ, holding only 160 cc. of water), in others being greatly dilated and capable of holding more than 2000 cc. of water.

The surface of the mucous membrane in a few cases shows other obvious gross changes. Over the pyloric area, it may be raised into small irregular projections, described by Louis (1826) and called by him *état mamellonné;* in other parts small cysts may be found, and a few areas may be seen, yellowish in colour and soft, which mark the parts where there is fatty degeneration of the glands. Superficial erosions are sometimes seen, as well as the smooth scars with no puckering, which they leave on healing.

Fibrosis of the Mucous Membrane—Cirrhosis and État Mamellonné.—Fibrosis of the mucous membrane is to a greater or less extent present in most cases of chronic degeneration of the mucous membrane secondary to inflammation. Those cases in which the change is so great as to be the most prominent condition are called cirrhosis, the pathological change being in all essential particulars similar to that in cirrhosis of the liver (Chapter XIV. p. 392).

The extent to which the mucous membrane undergoes fibrosis varies greatly ; in advanced cases the whole extent is involved as well as the other coats of the organ. These advanced cases of cirrhosis are rare (*loc. cit.*). In other cases, there is a lesser degree of fibrosis throughout the whole or the greater part of the mucous membrane, as well as in the submucous coat, the muscular tissue not being involved. In still others, the fibrosis is limited to certain areas, most commonly at the pyloric portion of the organ (as in the *état mamellonné*), but also in the mucous membrane of the cardiac region. These variations in the degree of fibrosis are of some importance, the lesser degrees being much more common than the advanced.

The *microscopical appearances* show that in the early stage there is a small-celled infiltration between the glands, well-marked near the surface of the mucous membrane which is denuded of epithelium (Fig. 26). In the later stages, the small-celled infiltration in part becomes fibrous tissue, which in contracting presses upon the glands. The glands atrophy, and either disappear completely or form cysts which are lined by flattened epithelial cells or contain granular *débris.* There may therefore be parts of the mucous membrane in which no glands are seen, or only the remains of the glands, or cysts (Figs. 22 and 27).

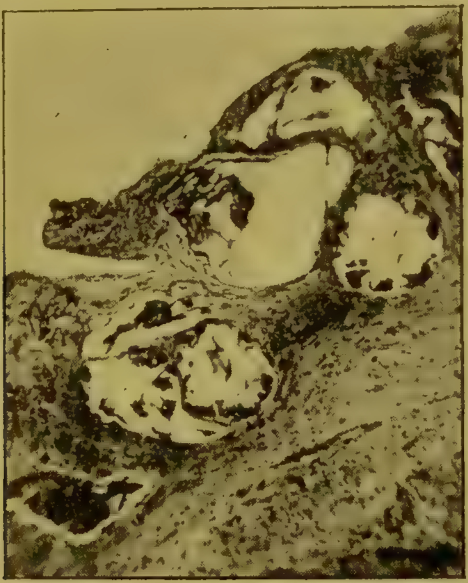

FIG. 22.—Cyst formation in the mucous membrane of the stomach. From a photograph, × 75. The specimen was taken from a female of middle age who died of pneumonia. Many parts of the mucous membrane were normal, but at the part shown in the figure, there was fibrosis with dilatation of the alveoli, resulting in the formation of cysts. The contents of the cysts were the degenerating cells of the glands: the submucous coat was thickened. From a preparation hardened in Müller's fluid and stained with logwood.

The fibroid change may affect the muscularis mucosæ, causing it to be greatly thickened, as well as the submucous tissue. The muscular coat is not infiltrated.

Two conditions may be treated separately as connected with this fibrosis of the gastric mucous membrane, viz. *état mamellonné* and the fibrosis that occurs in carcinoma ventriculi.

État Mamellonné (*Polyposis Ventriculi*) (Fig. 23).—This is a condition which is not very common, but has not infrequently been noted as a sequence not only of chronic inflammation of the mucous membrane of various origin, but of direct injury such as that produced by irritants and corrosives, ending in chronic inflammation. The pyloric portion of the stomach is most commonly affected, and presents varying appearances. One part of the mucous membrane may be quite normal,

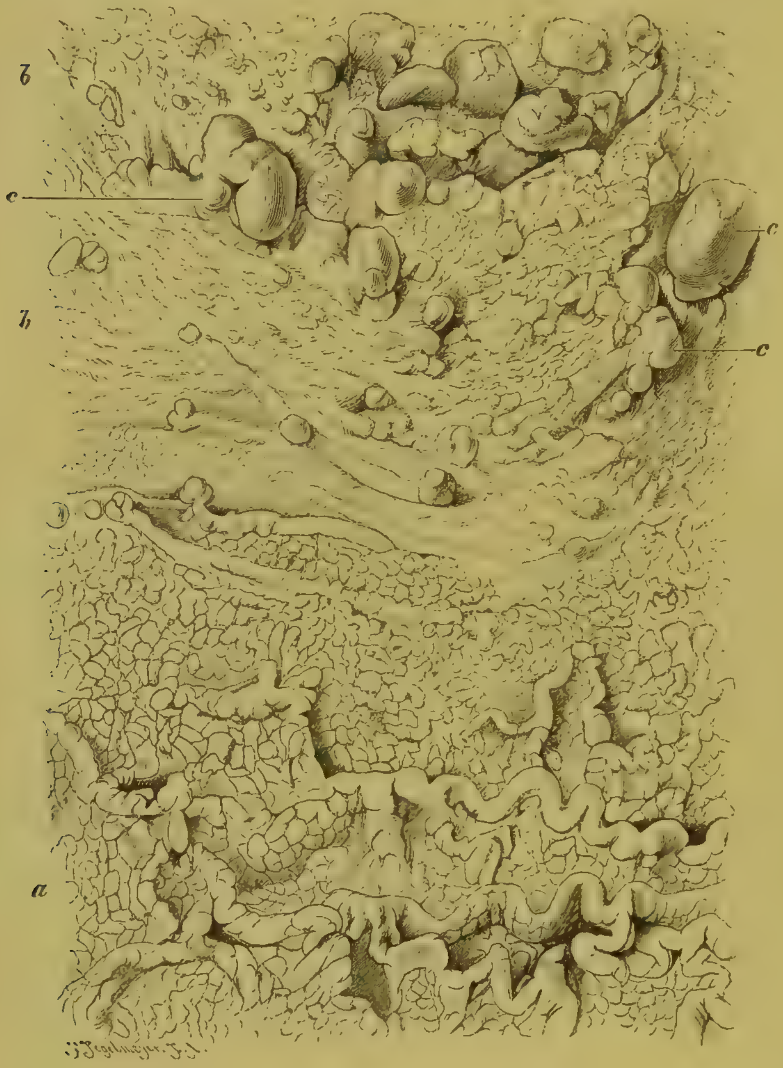

Fig. 23.—Atrophy of the mucous membrane of the stomach with polyposis. *a*, mucous membrane, normal in appearance ; *b*, smooth and atrophied mucous membrane ; *c*, polypi. (Ziegler.)

and adjoining it an area may be seen which is smooth, thin, and atrophied, while at another part there are either tough bands along the mucous membrane or projections, "papillæ or polypi," varying in diameter from one to two lines to a quarter or half an inch. Microscopically, the cause of these appearances is seen to be an increase of connective tissue in the mucous membrane, which by contracting has separated certain parts, and, constricting the base, raised them above the surface in the form of fungoid growths. The submucosa is also fibrosed, and the muscular tissue may also be in the same condition. The secretory glands also are compressed; their cells become degenerated and in some parts the glands are cystic.

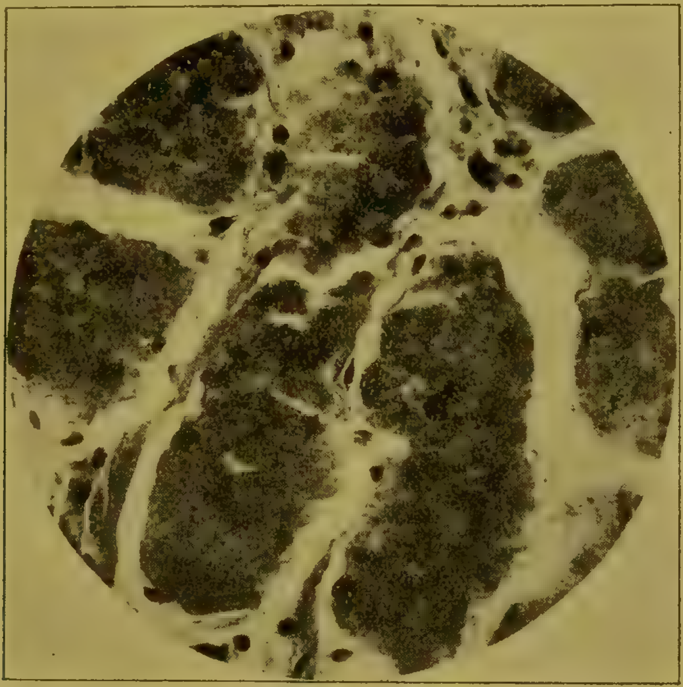

FIG. 24.—Gastric catarrh: fatty degeneration of the glands, early stage. From a photograph, × 350. From a case of pulmonary tuberculosis. Figs. 24, 25, and 26 represent different parts of the same stomach. The gastric glands are swollen, and the outlines of the cells are completely lost; the nuclei have disappeared in parts, and the cells are more granular than normal, fatty granules being also seen. The stroma is normal. From a preparation hardened in Marchi's fluid, and stained with logwood.

Fibrosis in Carcinoma Ventriculi.—In cases of cancer of the pylorus, even when the growth is quite limited in extent, there is usually a considerable degree of fibrosis near it, not only in the mucosa, but in all the thickness of the walls of the organ. This infiltration may be in great part cancerous in nature, but in other cases it spreads from the pylorus throughout the whole of the organ, and is then undoubtedly a condition of cirrhosis without cancer infiltration, and is associated with hypertrophy of the muscular coat. The naked-eye appearances of the organ in this condition are very similar to those in the stomach, the seat of a non-cancerous cirrhosis. This subject is more fully treated under "Cirrhosis Ventriculi" (Chapter XIV.), and "Cancer of the Stomach" (Chapter XVI. p. 468).

2. *Parenchymatous Degeneration of the Gland Tissue of the Stomach.*—The degeneration of the secretory glands by compression by an interstitial small-celled infiltration and fibrosis, which has just been under discussion, is a different process to a primary degeneration of the glands. In the first, the atrophy of the gland cells results from the interstitial infiltration; in the second, although there may be interstitial infiltration, the degeneration of the gland cells is a primary condition.

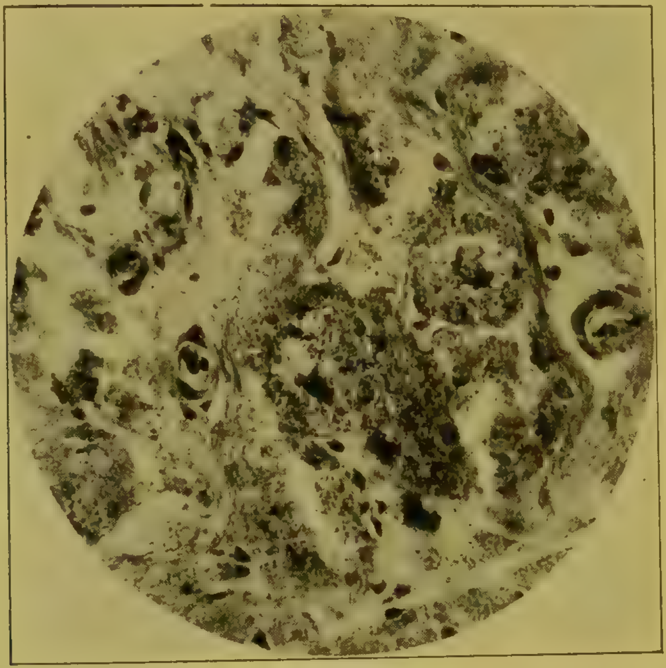

FIG. 25.—Gastric catarrh: fatty degeneration of the glands. From a photograph, × 350. From a case of pulmonary tuberculosis. There is no increase of fibrous tissue in the mucous membrane, but the gastric glands are seen in an advanced stage of fatty degeneration. Nearly all the cells have lost their outline; the nuclei are indistinct or have disappeared, and the protoplasm of the cell has been transformed into fatty granules. From a preparation hardened in Marchi's fluid, and stained with logwood.

More than once already it has been stated that in acute catarrh the cells of the secretory glands may undergo two forms of degeneration—a mucin degeneration and a fatty degeneration. Both these processes occur in chronic catarrh, and may, indeed, in rare cases, be well marked, and it is chiefly these degenerations which lead to the formation of cysts. The changes occurring in the glands in gastric catarrh are illustrated in Figs. 24, 25, and 26. In the early stage (Fig. 24), the glands are swollen, the cells are indistinctly outlined, the nuclei are disappearing, and the protoplasm of the cell is more granular, and fat globules are seen. When advanced (Fig. 25), the cells are completely destroyed by fatty degeneration, their nuclei have disappeared, and the fat granules are discharged into the lumen of the gland. The interstitial tissue may also become fatty. The whole of the gland may be thus affected from its orifice to its deepest part, and in some, although rare, instances, the deep part of the gland dilates, being constricted above, and forms a cyst. In some parts of the mucous membrane (Fig. 26) the gland tissue has disappeared,

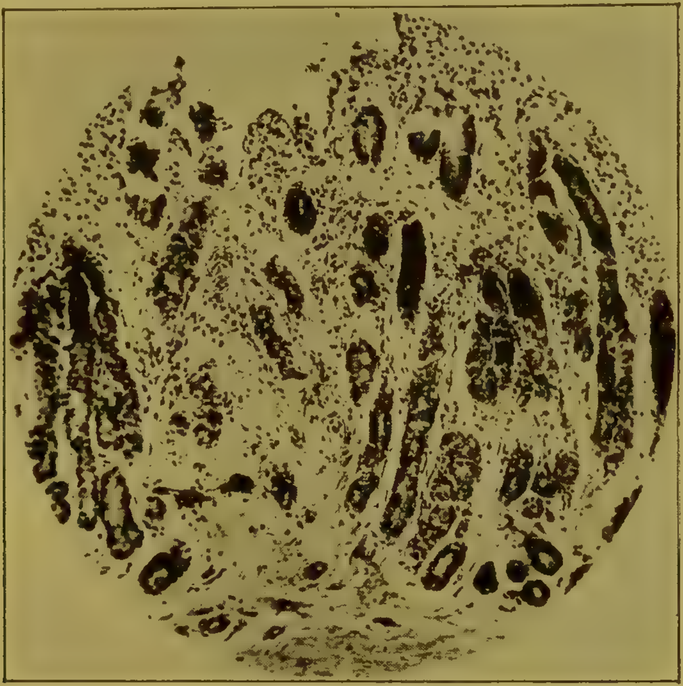

FIG. 26.—Fibrosis in gastric catarrh. From a photograph, × 70. From a case of catarrh in pulmonary tuberculosis. The gastric glands are in a state of atrophy, and are widely separated by recently-formed connective tissue, rich in cells. The mucous membrane had lost its epithelial lining. The other coats of the organ were normal. From a preparation hardened in Marchi's fluid, and stained with logwood.

and there is an increase in the interstitial connective tissue. All the changes shown in the figures are to be observed in one or other part of the same stomach.

A rarer condition than this is one in which the degeneration of the glands is mucinoid and not fatty, the change affecting the greater part of the mucous membrane. It is well illustrated by a case described by R. Langerhans,[1] observed in the Charité at Berlin. It was that of a woman aged fifty-eight years, who had no symptoms specially referable to the stomach during life,

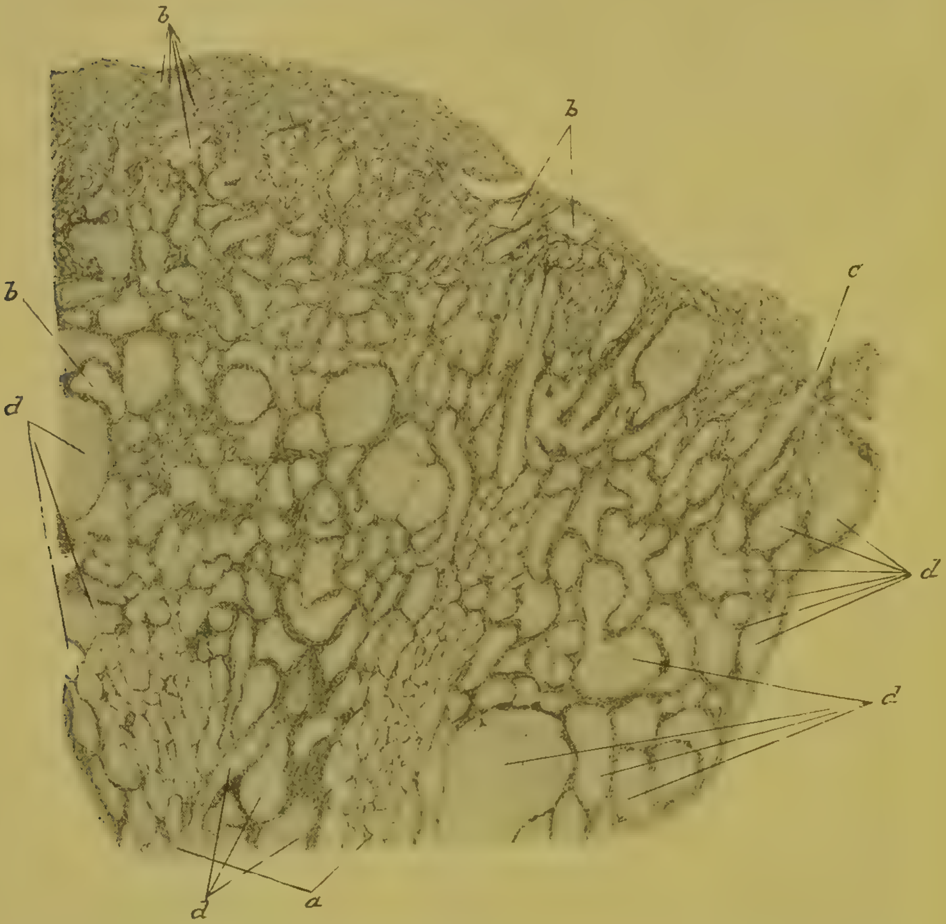

FIG. 27.—Mucoid and cystic degeneration of the gastric mucous membrane in a case of chronic tuberculosis, × 25. *a*, small cysts, not lined with epithelium; *b*, blood-vessels; *c*, gastric gland opening on the surface; *d*, medium-sized cysts, for the most part lined with epithelium, chiefly composed of goblet cells. At the surface of the mucous membrane there is a great increase of the interstitial tissue. (R. Langerhans.)

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[1] "Ein Fall von Gastritis catarrhalis chronica cystica prolifera," *Virchow's Archiv*, 1889, Bd. 116, p. 468.

and died of very chronic tuberculosis, affecting the lungs, pleura, peritoneum, and intestine. There was also atrophy of the kidneys and pancreas. The mucous membrane of the stomach was covered with thick mucus. The walls were generally and greatly thickened, and the mucous membrane, which was of a pale reddish-gray colour and soft and friable, showed the presence of bands of warty (polypoid) projections in parts, as well as small cysts. Microscopically, under a low power, as is represented in Fig. 27, the mucous membrane had the appearance of a large sponge, the glands being lengthened, dilated, and fused together, and in many parts transformed into closed cysts of varying size. Under a higher power, the glands could still be seen to be lined by epithelial cells, as the nuclei were still visible, but the bodies of the cells had undergone a mucinoid degeneration, and were in great part fused together. There was, in fact, in no part of the stomach any normal gland tissue remaining; the least abnormal was along the lesser curvature. In part the interstitial connective tissue was increased (hyperplasia); in others it was atrophied, as had previously been noted by Virchow in a case of cystic formation in chronic dysentery in which there were cysts produced by the fusion of glands.

This case has been quoted, although the degeneration of the mucous membrane was very advanced and occurred in a severe case of disease (tuberculosis), because it serves as an indication of the anatomical changes in some of the milder cases of chronic catarrh of the stomach, where there is a continuous secretion of an excess of mucus.

Anatomical Changes in the Muscular Coat of the Stomach.—The muscular coat undergoes degeneration under certain conditions, the chief of which is dilatation of the organ. In cachectic conditions degeneration is also noticed. There may either be simply atrophy of the muscle cell or fatty degeneration. A colloid degeneration of the muscle has also been described,[1] but this is not common. Finally, where there is general fibrosis of the stomach wall, the muscle cells are atrophied by compression (Chapter XIV.).

Pathological Condition and Process of Digestion.—In gastric catarrh there is great interference with the normal blood supply to the organ during digestion. In the acute forms there is stasis to a greater or less extent, and therefore almost complete abeyance of the stomach functions, both secretion and motor activity being affected. In the less acute forms and in the chronic, there is great diminution in the amount of hydrochloric acid secreted, pepsin being present; the secretion of pepsin is affected in long-standing cases. In the early stages, and at times during the chronic catarrh, there is frequently motor irritability of the organ; but the tendency in chronic catarrh is towards diminution of motor activity and towards dilatation of the organ. Absorption in acute and chronic catarrh is greatly affected, to some extent by the secretion of mucus, but chiefly by the condition of the mucous membrane.

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[1] R. Maier, quoted by Ziegler, "Lehrbuch der pathol. Anatomie," vol. ii., 1887.

The process of digestion is greatly modified by these changes. In acute catarrh there is practically no digestion, the food remaining unacted upon in the organ (see p. 101); but in milder forms of catarrh and in the chronic form, there is inability to digest an ordinary mixed meal, owing chiefly to the diminished acidity of the gastric contents, and to weakness of the movements, but also to the presence of mucus which mechanically interferes with digestion. Moreover, even if the stomach does secrete some effective gastric juice and is capable of some peristalsis, it soon ceases work, *i.e.* although it may begin digestion it cannot complete it. A liquid therefore remains in the stomach which is slightly acid, neutral, or alkaline (see p. 113), and contains peptones, salts, and mucus; it is usually vomited. Delay of food in the organ is therefore present, and due not only to the diminished chemical processes, but to a diminished motor activity. It is much more marked, when, as the result of prolonged catarrh, or of a series of subacute exacerbations of the disease due to dietetic irregularities, the organ dilates. It dilates only to a moderate extent, as a rule, and bacterial fermentation of the food may ensue and increase the mischief, leading to hyperacidity of the stomach contents due to organic acids, and to a continued irritation of the organ. Without dilatation of the organ, bacterial fermentation may occur, owing to the diminished quantity of hydrochloric acid secreted.

SYMPTOMS.

1. *Acute Catarrh.*—Acute catarrh of the stomach is a disease of sudden onset, although it may be preceded for a few days or longer by some slight signs of indigestion of food. Its immediate onset is usually associated with some indiscretion of diet; such as a large and indigestible meal—a food debauch, or an excess of alcohol—a drink debauch. In some cases a sudden emotion appears, with these causes, to be an auxiliary factor. The actual onset is characterised either by vomiting preceded by great epigastric pain, or by vomiting associated with a rigor and a slight rise of temperature to about 100° F. The fever is, as a rule, of but short duration, not lasting as long as the gastric symptoms. The patient suffering from acute gastric catarrh, soon after its onset, is seized with great prostration, so that no bodily or mental exertion is possible. He takes to his bed; the face is pale, sometimes drawn and sunken; the skin is cold and clammy, and is sometimes slightly covered, especially on the face and upper part of the body, with a clammy sweat. The pulse is usually rapid, regular, and compressible. There may be diffuse pains in the back and limbs, and giddiness on exertion is sometimes a well-marked symptom. Mental symptoms are not common, although slight delirium has been observed. There is complete loss of appetite, indeed, often a loathing of food, accompanied by thirst and a dryness of the mouth, which are not infrequently very distressing. Herpes labialis is not infrequently present. The patient, indeed, in the early stage of gastric catarrh, shows the symptoms of acute irritant poisoning; it is, indeed, acute irritation of the stomach, ending in inflammation.

The symptoms referable to the gastro-intestinal tract need more careful consideration.

The tongue is dry and thickly coated with a white or yellow fur. Diarrhœa may be present in the early stage of acute catarrh, being due to the irritant food taken, but is usually succeeded by constipation, which is a well-marked and characteristic symptom during the course of the disease.

Pain in the stomach region is a feature of most cases of acute catarrh, although in some few cases it may be absent. It is a burning pain often darting in character, passing through to the back and is accompanied by pain and a sense of oppression in the chest. There is well-marked deep tenderness all over the stomach region. The abdominal muscles are contracted over the organ in the early stages; later in the disease they relax. The tenderness of the stomach is associated with quiescence of the diaphragm, so that the respiration is costal.

Vomiting is another feature of acute catarrh. As has been said, it sometimes initiates the attack and it may be frequently repeated, so frequently as to be a very distressing and painful symptom. Even after the stomach is emptied, ineffectual retching, sometimes associated with painful hiccup, is observed. Any attempt at feeding even with milk is succeeded by a rejection of the food and violent retching. The vomiting is in some cases a less marked symptom; during the whole course of the disease vomiting may occur only three or four times. The *vomited matters* consist, at the onset, of partly digested food; but later the food shows no sign of digestion. In cases where there is not excessive vomiting the food may remain for a long period in the stomach, showing on removal no sign of having been acted upon by the gastric juice. The reaction is slightly acid, neutral, or more commonly faintly alkaline. As a rule tests for hydrochloric acid give negative results, and peptones may not be discovered. In some cases the vomit is very acid, owing to the presence of organic acids—butyric, lactic, and acetic acids. This sign of fermentation of food is, however, a later sign of the catarrh, and not an early one, unless it has existed before the acute attack supervened. Slimy mucus is present. Streaks of blood due to the violent retching may be present in the vomit; but as a rule any large quantity of blood is not brought up. Bile may be present, but only at the end of a long period of vomiting.

The urine is scanty, of high colour and specific gravity, and deposits lithates; but does not usually contain either blood or albumin.

The disease runs a fairly rapid course, convalescence being established in three days or a week. Frequently, however, the acute catarrh passes on to a subacute or a chronic. The general symptoms first diminish, the pulse becomes less rapid, the feeling of prostration passes away, and the vomiting diminishes as well as the epigastric pain. The appetite as a rule is diminished for a long time, and the foul odour of the breath is often persistent.

When acute gastritis supervenes in the course of an acute disease, its symptoms are often not so characteristic as the foregoing. Those referable to the stomach are, however, practically the same, viz. the vomiting of food partly digested, but little free hydrochloric acid being present; epigastric pain and diffuse tenderness are also observed.

Prognosis.—Acute gastric catarrh is not a common disease; not infrequently acute gastric irritation (p. 196) and severe bilious attacks have been mistaken for it. The prognosis is good, especially in adults, although it may end in chronic catarrh. When complicating acute disease, such as scarlet fever, it is a serious condition, and during convalescence its onset seriously delays recovery.

Diagnosis.—Acute gastric catarrh is a disease in which the symptoms are chiefly referable to the stomach, viz. the vomiting and epigastric pain and tenderness. The absence of hydrochloric acid and of pepsin and the presence of mucus in the vomit are diagnostic points. At its onset it may be mistaken for toxic gastritis (Chapter IX.), or even for typhoid fever in its early stage. But in gastric catarrh, fever may be absent; it is never high in adults, and soon passes away. Moreover, its acute symptoms soon pass off, and the spleen is not enlarged.

Treatment.—The treatment of acute gastric catarrh is like that of inflammatory conditions elsewhere, to give the organ rest. As little food is to be given as possible and as little medicine. As regards food, complete abstinence is the best treatment during the first twenty-four or thirty-six hours in the severer forms, rectal feeding being adopted (Chapter XI. p. 342). Thirst may be relieved by a limited allowance of ice, by the use of effervescent lozenges, or by allowing the patient to sip small quantities of an acid lemonade (see Chapter X.).

The stomach must be kept practically empty to allow the inflammation to subside; if it has not been emptied by vomiting, as will be shown by the bringing up of small quantities of liquid, or if the vomiting is otherwise ineffectual, it is best to empty the organ by washing it out with a dilute solution of sodium bicarbonate, the soft stomach tube being used (see Chapter XII. p. 359).

Excessive vomiting followed by violent retching in which nothing is brought up from the stomach is best treated by a hypodermic injection of morphine. Two minims of the injectio morphinæ hypodermicæ may be injected, to be followed in two hours by a second injection of the same quantity. As a rule a further repetition is unnecessary to relieve the vomiting. In excessive epigastric pain, morphine may also be used hypodermically, but again with caution and not until local applications have been found of no avail in affording relief. Hot belladonna fomentations over the upper part of the abdomen frequently relieves, glycerinum belladonnæ (1 in 4) being first painted over the skin and a flannel wrung out in boiling water placed over with oiled silk and cotton wool. A light bandage over this gives support and quiescence to the organs, and hence is an aid in the relief of pain. In some cases, a cold pad over the epigastrium relieves; either a light ice-bag (with finely powdered ice) or a four-folded piece of flannel wrung out in cold water and frequently changed.

Constipation is best treated for the time by enemata of about one pint of soap and water, or of thin and strained oatmeal; half an ounce of castor-oil may be added to the oatmeal enema (Chapter X.).

Medicinal treatment is of great use when the acute stage of the disease is passing away. Acids after food or with food are the best remedies. Acidum hydrochloricum dilutum or acidum nitro-hydrochloricum dilutum may be given in 10 to 15 minim doses. If there is still epigastric uneasiness or pain, to the acid mixture vii.–x. minims of liquor morphinæ hydrochloratis or tinctura cannabis indicæ may be added (Chapter X. p. 300). The later treatment is one of careful diet and of general tonics.

2. *Chronic Catarrh.*—Some difficulty presents itself in the discussion of the symptoms of chronic gastric catarrh. The frequency of this affection has been variously stated. By some it has been considered to be the condition present in nearly all cases of chronic dyspepsia, by others it is considered as rare. It is a less common affection than most have supposed. There is no evidence that the cases described as gastric irritation are in any way of the nature of inflammation of the mucous membrane; there may be a slight excess of mucus in response to a great irritation at one particular time, yet there is not that continuous excessive secretion of mucus or that diminution of functional activity, which is characteristic of a catarrhal inflammation of the stomach. Again, what has been described as acute gastric irritation (p. 196) is considered by some as acute simple gastritis, but this view is decidedly wrong. The rare affection acute gastritis is a much more severe one than acute gastric irritation. On the other hand, it has been fully dwelt upon in discussing the etiology of gastric irritation and of gastric catarrh, that the factors in their causation are mainly the same; catarrh is, however, an inflammation, irritation is only a passing change in the stomach. In actual practice, cases run on into one another, and it is often difficult to tell where gastric irritation ends and catarrh begins, the early stage of inflammation being sometimes associated with hyperacidity (" acid " catarrh).

Catarrh when once established is a serious affection, and is characterised clinically by a chronic afebrile course, with subacute exacerbations, by epigastric pain and diffuse tenderness, by the vomiting of mucus and of a liquid greatly deficient in hydrochloric acid.

Onset.—Its onset is frequently insidious, following a long course of gastric irritation, or it may be sudden, following on acute gastritis or arising in the convalescence of acute fevers, etc. (see Etiology, p. 222).

When insidious in origin, it presents the train of symptoms which have been described as characteristic of gastric irritation. These need not be repeated. When developed, the symptoms, although general, are mainly referable directly to the stomach and stomach region, being related to the ingestion of food and to the delay of undigested food in the organ.

Epigastric pain, or pain over the whole stomach region, begins directly after the ingestion of food and lasts a varying time, sometimes for an hour or two, sometimes until the next meal. It is sometimes severe, and is only relieved by emptying the stomach. This occurs either by spontaneous vomiting or by the patient exciting vomiting by putting the fingers down the throat. So severe is the epigastric pain and distress in many cases, that the patient, although he takes food, more from duty than inclination, yet before the next meal always excites vomiting, as he is unable to endure the distress. He may indeed half starve himself owing to fear of this distress. This occurs chiefly in subacute catarrh or in chronic catarrh aggravated by neglect. In milder cases the epigastric pain does not come on immediately after food, although there may be a sense of fulness and discomfort directly after ingestion, but the pain comes on in an hour or two and is then continuous for some time. Sometimes vomiting ensues and relief is obtained, but the pain may gradually disappear without vomiting being produced. The pain is not confined to one particular spot in the stomach region; it is of a burning character, diffuse, and may go through to the back.

The *epigastric tenderness*, with which the pain is associated, is sufficiently characteristic. At one time or another it is discoverable in all cases of chronic catarrh, and is rarely absent in subacute cases. It is diffuse over the stomach region, not excessive even on deep palpation; but certain areas may be more tender than others. It is in many cases deep tenderness and not superficial. The following diagram (Fig. 28), from a case of chronic catarrh of over twelve months' duration in a young married woman of twenty-five years, illustrates this diffuse tenderness over the stomach region. In this case there was probably an erosion of the mucous membrane, as a slight hæmatemesis had occurred.

In milder cases, the tenderness may be slight and limited to one and two areas in the stomach region, below the left hypochondrium or in the upper epigastrium, as in the accompanying diagram (Fig. 29), which shows the areas of tenderness in the stomach region of a woman aged thirty-four years, who dated all her gastric symptoms from an attack of rheumatic fever five years previously. There was dilatation of the stomach.

A feature of great importance in regard to this epigastric tenderness is that it disappears with remarkable readiness under suitable treatment, a distinction of great value from the epigastric tenderness due to ulcer of the stomach.

Variations in these symptoms are met with in some cases of catarrh. There may be epigastric pain after eating, but no tenderness, and both epigastric pain and tenderness may be absent. It is not an infrequent occurrence in such cases for no tenderness to be discovered and the patient disclaims any epigastric pain after food. It will be found, however, on close examination that not long previously this symptom has been present, but that owing to treatment (change of diet chiefly) it has become less or has disappeared. Indeed, epigastric pain and tenderness are present chiefly in the

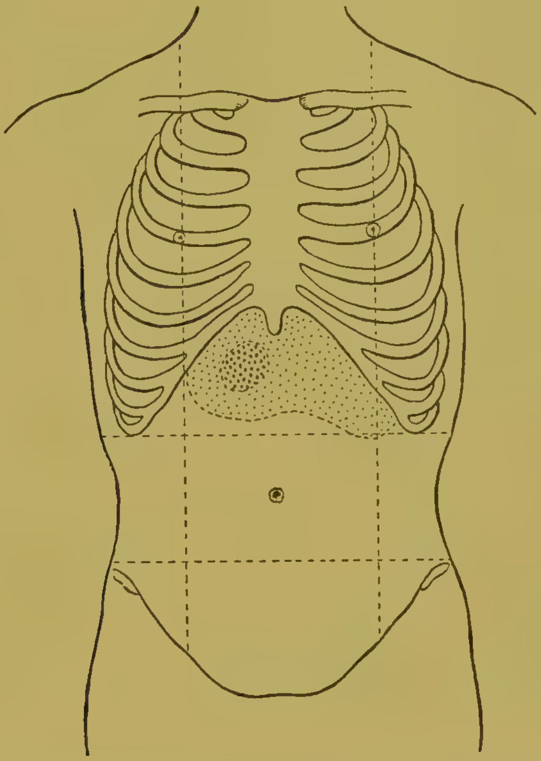

FIG. 28.—Area of tenderness in the stomach region in chronic gastric catarrh (subacute exacerbation). The larger area is that of slight tenderness: over the smaller area, the tenderness was more marked. Female, aged twenty-five years; duration of illness, fifteen months.

subjects of neglected catarrh, in those who in spite of advice will take unsuitable food, which keeps up and aggravates the disease.

Reflex pain in the chest and between the shoulders is also met with, as in cases of gastric irritation.

Nausea is a frequent symptom. It usually precedes vomiting, but exists when no vomiting occurs. The time of onset of the nausea varies; it may come on directly after food has been taken, but is more commonly delayed until one or two hours after a meal. When it comes on soon after a meal it is so distressing as to prevent the patient from eating any more food, but the nausea later developed may be only a momentary symptom passing off without vomiting.

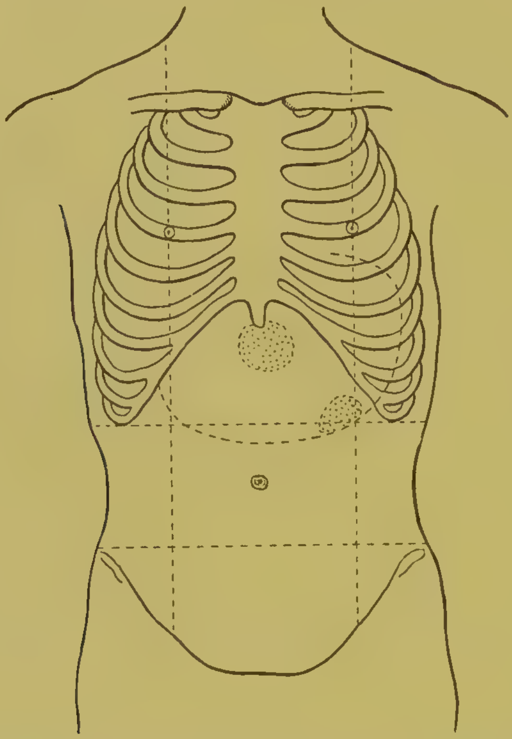

FIG. 29.—Areas of tenderness in chronic gastric catarrh, with dilatation of the stomach, following rheumatic fever. Female, aged thirty-four years; duration of illness, five years.

Vomiting is a symptom never absent in the course of cases of chronic catarrh. It varies in intensity. A not uncommon history is for the patient to have attacks of vomiting at varying intervals during a long period, it may be six months, a year, or longer. During this time there are periods when the vomiting is more frequent, perhaps once a day or once in two days, and it may eventually occur after every meal or be excited by the patient himself. It is often during those attacks or subacute exacerbations of the disease that the patient seeks medical relief. The vomiting is in relation to food, it occurs after a meal, and is frequently to be ascribed to some particular indiscretion in diet. In very frequent vomiting it occurs almost directly after a meal, but more usually at an interval of half an hour to two hours. The vomiting act when complete is not usually succeeded by much retching, but gives relief to the symptoms produced by the meal. Morning vomiting may occur, both in alcoholic and non-alcoholic subjects.

Even when vomiting is not a prominent symptom, eructations of small quantities of liquid and of gas are common, due to the irritability of the stomach in the presence of slowly digesting or of undigested food.

Character of the Vomited Matters.—The vomited matters in chronic gastric catarrh vary somewhat in composition. Their chief characteristics are that they contain unpigmented mucus, and a deficiency of hydrochloric acid. A typical example of vomited matters in this condition has already been discussed (p. 141). The mucus varies in amount; in some cases it is so abundant that the vomited matters are exceedingly tenacious, and cannot readily be poured from one vessel to another, a little liquid being enclosed between the masses of mucus. In other cases it is much less in amount, consisting only of a few pellets of glairy mucus floating in the liquid. It may be absent from the vomit, even in cases where some mucus is obtainable by washing out the stomach. In the later stages of chronic catarrh, when there is atrophy, mucus may be absent both in the vomit and in the stomach; mucin is no longer produced by the goblet cells of the mucous membrane. Microscopically, besides the strings of mucus, a few leucocytes are seen, and in many specimens goblet cells of the mucous membrane, showing a greater or less degree of degeneration. Particles of food (coagula of milk, etc.) are present, and there may be bacteria. Red blood corpuscles are sometimes seen, even when there is no naked-eye evidence of blood. Mucus may be found in some instances in the vomit, and yet not come from a catarrhal stomach. This occurs in cases of pulmonary tuberculosis and bronchitis, in which the sputum is swallowed. The pigmentation of the mucus, and the nummulated character, as well as the large number of pus-cells present, giving the mucus a yellow or greenish colour, will serve to distinguish swallowed sputum from the mucus in cases of gastric catarrh.

The *reaction* of the vomit may be slightly acid, neutral, or faintly alkaline; when there is bacterial fermentation, it may be strongly acid, but this is rare. The diminished acidity of the gastric contents may be in part due to the excess of alkaline mucus present; but in some cases mucus is absent, and the liquid still be alkaline. Tests applied to the liquid (see p. 125 and 141) show that there is a great diminution in the amount of free acid present, and that free hydrochloric acid may be absent. Cases of catarrh which show a large degree of acidity of the vomit are always cases where, from bacterial fermentation in a dilated stomach, organic acids (lactic, butyric, acetic) are formed in excess. Catarrh in a dilated stomach is not an uncommon event.

The *presence of digestive ferments* in the vomit is determined in the manner previously described (Chapter V.). In mild cases, and in those of not long-standing, as well as in the convalescent stage of the acuter forms of the disease, pepsin is found, and is active, as in the case recorded previously (p. 141), and pepsin may be present even although the hydrochloric acid is greatly diminished in quantity. According to Boas, pepsin may disappear, and only pepsinogen be present, being brought into activity by the addition of hydrochloric acid. The curdling ferment appears to be the last to disappear in cases of catarrh (Boas).

In moderate cases, the products of digestion (albumoses and peptone) and a large quantity of undigested food may be found in the vomit a long time after a meal, even of the simplest kind. In the later stages of catarrh, and in subacute cases, there may be practically no sign of digestion of the food, which is vomited in the same condition as swallowed (p. 268).

The examination of the vomited matters in cases of gastric catarrh is therefore all-important. Knowing the character of the previous meal, and the time elapsing between the meal and vomiting, a fairly accurate estimate is obtained of the process of digestion in the organ, of the activity of the secretion of gastric juice, and of the degree of inflammation as estimated by the amount of mucus and of the hydrochloric acid.

Test-meals.—Similar information may be obtained by means of a test-meal (p. 149), and when there is no vomiting this method is to be employed to determine the degree of deterioration in the functions of the stomach. Ewald's test-breakfast or Leube's simple meat meal may be used, or one consisting of half a pint of boiled milk and the finely coagulated whites of two eggs (*loc. cit.*), the stomach contents being removed in one to two hours.

Hæmatemesis.—In gastric catarrh there is no profuse hæmatemesis; but not infrequently blood is present in the vomited matters, sometimes only in streaks, sometimes to the extent of two or three ounces. As a rule the blood is red, not discoloured or tarry. The hæmatemesis is not frequent, perhaps occurring once or twice in the course of the illness. When blood is present only in streaks, it is usually associated with violent retching, as in other conditions, *e.g.* the retching produced by violent and prolonged coughing, and that accompanying sea-sickness. In the larger degrees of hæmatemesis occurring in catarrh, an erosion of the mucous membrane is usually present, or the form of pitting described as occurring in mechanical congestion of the organ (see p. 229, and Fig. 19, p. 230). Erosions are not uncommon in gastric catarrh, but they rarely lead to profuse hæmatemesis; and when a profuse hæmatemesis occurs in the course of gastric catarrh it is indicative of ulcer of the stomach, or it may be of some disease of the body other than the stomach (see Hæmatemesis, Chapter XIII.).

Flatulence is a frequent symptom, and is due to one or other of these causes previously discussed (p. 161). When a prominent symptom, it usually occurs in association with dilatation of the organ, and the gas may either be regurgitated from the small intestine, or it may be formed in the stomach by bacterial fermentation of the food, an occurrence predisposed to by the deficiency of hydrochloric acid. The degree of flatulence varies greatly in different cases, but it is not commonly the chief symptom complained of.

Dilatation of the stomach is frequently present. Its signs and symptoms are discussed in Chapter XV.

The symptoms directly referable to the stomach are of chief importance in gastric catarrh; but there are others which, in association with the stomach symptoms, are of value in the recognition of the disease.

In chronic gastric catarrh there is no fever, and the onset of fever is to be attributed to some other condition than that of the stomach.

The *face* is frequently pale, sallow, and of an earthy complexion, especially in subacute gastric catarrh. It is not usually expressive of pain, except during the distress caused by a meal, but tends to become somewhat anxious looking in cases of long-standing.

Appetite.—Although the appetite in chronic gastric catarrh is somewhat variable, it is usually diminished, and in cases of long-standing, as in the subacute exacerbations of the disease, it is greatly diminished, and there may be profound anorexia. In the early stage of the disease, the appetite is not materially affected, and in cases of "acid" catarrh it may even be increased for a time. In some cases there is a morbid craving for unsuitable and irritating articles of diet. When atrophy of the mucous membrane is present the appetite is always diminished.

Thirst may be complained of in subacute catarrh, but it is usually absent in the chronic disease. When dilatation of the stomach is present it may also be observed.

The *tongue* in chronic gastric catarrh is usually coated with a thick, white, or whitish-yellow fur, and the mouth has a nasty clammy taste; the breath is often offensive. The degree of coating of the tongue varies, and is more or less dependent on the character of the food taken, and the delay of food in the organ. The tongue may be broad and flabby, as in those cases which arise in the course of gastric insufficiency, but usually, and especially in children, it shows the signs of irritation in enlargement and reddening of the fungiform papillæ, especially near the tip ("strawberry-tip"). The fœtor of the breath is due partly to particular articles of diet taken, and in some cases to bacterial decomposition of the remains of the food in the stomach, but in the majority of cases it is to be ascribed to the condition of the mouth itself; to the fur on the tongue and to carious teeth.

The *bowels* are usually constipated, being opened once in three or seven days or longer, especially in women, or opened only when aperient medicine is taken. Frequently mucus is present in the stools, due to the mucus from the stomach, passing, as it usually does, undigested through the intestinal tract. The amount of mucus passed in this way in subacute cases may be very great, long cylinders over two feet in length being found in the motions (p. 269). Diarrhœa may be present, due either to the retention of scybala in the colon or to particular indiscretions of diet.

Urine.—In cases of subacute catarrh, and especially in cases of chronic catarrh which have been neglected and aggravated by dietetic indiscretions, the urine is passed in small amount, and is not infrequently neutral or alkaline and cloudy from a deposit of phosphates. No albumin is present as a rule. That this condition of urine is due to the stomach condition is shown by the effect of treatment. By the prescription of a simple and suitable diet and of acids after meals, the urine increases in quantity, becomes acid, and although it may for a time show an excess of phosphates by the boiling test, this soon disappears, and it then does not show any difference from the normal. In some of these cases, however, there is a continued diminution in the excretion of chlorides as in the case of gastric irritation (p. 118).

Nutrition of the Body.—In the majority of cases of chronic catarrh there is wasting, which is well-marked if the condition is subacute or if there are subacute exacerbations. The wasting may be continuous if the disease is neglected or the treatment injudicious, but it has this distinguishing feature, that suitable treatment soon enables the patient to regain weight. Indeed, with many cases of catarrh, the following history is a very common one: a more or less steady fall in weight, then by suitable dietetic and medicinal treatment a recovery of the weight; the patient now feeling better commits dietetic indiscretions and a loss of weight ensues, which is regained by treatment. So the condition goes on.

Repeated vomiting in the subacute attacks, and the voluntary starvation adopted by the patient as a means of relief, also aid in some cases the loss of weight. Catarrh of the small intestine is not infrequently associated with gastric catarrh, and exercises a profound influence not only on the process of digestion in the small intestine, but on the absorption of the digested products. In such cases, wasting may for a time be a well-marked symptom. In gastric catarrh, supervening in the course of chronic pulmonary tuberculosis and chronic Bright's disease, especially the granular contracted kidney, wasting is a prominent symptom, and in chronic Bright's disease the gastric condition may almost completely mask the renal, which is the more serious.

Nervous symptoms, which have already been fully discussed (Chapters VI. and VII.) may be associated with chronic gastric catarrh. Besides suffering from vertigo, patients not infrequently get into a desponding, often melancholic condition.

Course and Duration.—The course of uncomplicated chronic gastric catarrh is a very varying one. The most usual course is for a patient to have symptoms for a certain period, six or eight months, and then by treatment to have an intermission, during which time by care in the diet and the mode of living, there is more or less complete relief; but a return of the symptoms is very apt to occur, it may be after a year or several years' intermission, and is to be ascribed either to a particular dietetic indiscretion (alcoholic drinks, tea, or large and unsuitable meals) or to a change of the mode of living, as in going from one country to another, or removing from the country to town; precisely as in gastric irritation. In the young, the course of uncomplicated gastric catarrh depends almost solely on the treatment and on the care with which the patient keeps within the range of his digestive possibilities. In the middle-aged and in the old, on the other hand, chronic gastric catarrh runs a much longer course, owing to its being less amenable to treatment. In gastric catarrh complicated by tuberculosis or by chronic Bright's disease, the course is frequently a prolonged one, and is apt, even if held in check by appropriate treatment, to lead to permanent gastric insufficiency, due to degeneration of the mucous membrane.

This may also result from uncomplicated gastric catarrh, and a condition of what may be called atrophic gastric catarrh ensues, for which there is no cure. Such patients are permanent invalids as regards their digestion, and have to live on a strictly regulated diet (Chapter XI., Permanent Gastric Insufficiency). It is a condition more common in the middle-aged and in the old than in the young (see also Atrophy, p. 280).

In subacute catarrh, the disease may last from onset to cure for six or twelve months, but it may terminate in a permanent injury to the digestive powers of the organ.

Prognosis.—The prognosis of uncomplicated subacute and chronic gastric catarrh in the young is good. It is the period of activity, with great resisting power of the organism to disease. In the middle-aged and old with settled habits and with a mode of life, difficult or perhaps impossible to change: in catarrh associated with pulmonary tuberculosis and chronic Bright's disease, the prognosis is not so good, and depends in great part on the results of initial treatment. For in such cases, although a course of treatment relieves the more severe symptoms and may remove them altogether, yet continuous observations have to be made in order to determine the degree of permanent gastric insufficiency. In such cases the prognosis is aided, and may indeed be made by means of the methods already described for determining the activity of the functions of the stomach.

Chronic or subacute gastric catarrh, associated with dilatation of the stomach, is a condition in which a guarded prognosis must always be given. In this case, again, the results of treatment will be frequently a reliable guide.

Gastric catarrh may also be associated with carcinoma of the stomach, and sometimes masks the primary disease. Such cases are difficult and complicated, and are discussed later (Chapter XVI.).

Diagnosis.—It will have been seen from the symptoms detailed of both affections that chronic gastric catarrh bears a close superficial resemblance to chronic gastric irritation; and their separation clinically might be considered as unnecessary. Yet since chronic gastric catarrh is an inflammatory process, cases of gastric irritation cannot be included in its range. By some, indeed, all such cases are called "dyspepsia," a name which does not help either clinically or pathologically. By others, the term dyspepsia being retained for a somewhat indefinite class of disorders, most of the cases described under the heading of gastric irritation are placed under that of chronic catarrh. Although it cannot be said that our knowledge, clinical and pathological, is complete of such cases, yet chronic gastric catarrh has certain features which the cases classed as gastric irritation do not possess. On the other hand, as has already been fully explained in discussing the etiology and symptoms of both conditions, it is evident that gastric irritation (which is purely a clinical classification of cases) may and does readily pass into catarrh or inflammation, and indeed what has been described as the permanent stage of gastric irritation is in many cases a stage of degeneration of the mucous membrane due to catarrh. It is difficult to be sure of this, and such cases present great difficulties in their pathological explanation. The advantage of separating gastric irritation and gastric catarrh clinically lies in the fact that cases of gastric irritation are much more amenable to treatment than cases of catarrh. Gastric catarrh is indeed a serious condition, and may lead to advanced degeneration of the mucous membrane of the stomach.

Chronic gastric catarrh is characterised by the following combination of symptoms:—

1. It is essentially a chronic affection, although it may have subacute exacerbations.

2. As regards the symptoms, epigastric pain during the time food is in the stomach and occasional vomiting are in many respects diagnostic.

3. The vomited matters are characteristic, showing a diminution or absence of hydrochloric acid, and the presence of mucus.

4. By means of a test-meal, if there is no vomiting, this diminution of hydrochloric acid and the presence of mucus may be demonstrated.

5. Owing to the weakness of the muscular wall, dilatation of the stomach is apt to supervene.

Actual pain in the epigastrium, following the ingestion of food, occurs in three diseases of the stomach—gastritis, gastric ulcer, and cancer. It is not present in gastric irritation, except as a very occasional event, and it is absent in gastric insufficiency; in both these conditions the feeling in the epigastrium is one of weight, of undue fulness and distention rather than actual pain. In gastric ulcer, uncomplicated by catarrh, the pain after food is localised, sometimes remarkably so, while in gastric catarrh it is diffused over the whole stomach region. In cancer of the organ the pain varies; in many cases it has no relation to food, but when occurring after a meal it is usually diffuse. In the fasting stomach in cancer, pain may be present; so that in many cases there is continuous epigastric pain. Epigastric tenderness is frequently of aid in the diagnosis. It is absent, as a rule, in gastric irritation and gastric insufficiency, but in the former it may be present as an occasional symptom after a meal; in that case it is diffuse. In gastric ulcer, the tenderness is often remarkably local, to the right and above the umbilicus or just below (1 or 1½ inch) the xiphisternal notch (Chapter XV.). In gastric catarrh, as has been pointed out, tenderness is sometimes absent even if there is pain after food, and when present it is diffused all over the stomach region, or is limited to certain areas (p. 252); it is never localised, and repeated examination never reveals the localisation of tenderness in one spot, as it does in gastric ulcer. In cancer, when a tumour is absent (the presence of a tumour is diagnostic, Chapter XVI.), the tenderness may be localised, and is frequently elicited over the pylorus, but it is not so localised as in ulcer.

The *vomiting* of gastric catarrh frequently resembles that of gastric irritation and, in subacute cases, that of gastric ulcer. The vomiting in cancer, as in gastric insufficiency (when it occurs), is more commonly of that kind observed in dilated stomach (Chapter XIV.). In gastric irritation, the vomiting can be distinguished from that of catarrh; it occurs after food, as in catarrh, but is a less frequent symptom than in the latter disease. In gastric ulcer, the vomiting is still more frequent than in gastric catarrh, and relieves the epigastric pain.

Of more diagnostic significance are the characters of the vomited matters. In catarrh, the characteristics of the vomit are the great diminution of the amount of hydrochloric acid and the presence of mucus. Mucus is absent from the vomit of gastric irritation and from that of ulcer and cancer, unless catarrh complicates these diseases. In gastric irritation and in ulcer, the hydrochloric acid may be in excess, in normal amount, or in the later stages of the disorder somewhat diminished. In cancer and great dilatation of the stomach, the vomit may contain a diminished quantity of hydrochloric acid, but it contains a large excess of organic acids, chiefly lactic. The characteristic of the vomit of gastric catarrh is the presence of mucus with a diminished percentage of hydrochloric acid, and in severe cases the presence of mucus in the stools, intestinal catarrh being absent.

Hæmatemesis is not a symptom which is present in gastric irritation or insufficiency. It may be present in catarrh, and is then usually slight in amount and not often repeated. When profuse in catarrh, it may be associated either with portal obstruction, as in cirrhosis of the liver, or in dilatation of the right side of the heart as in mitral disease, or, more rarely, in chronic Bright's disease. It may be the result of an erosion of the mucous membrane, and is then slight in amount. In ulcer the hæmatemesis is more profuse, and in cancer it occurs towards the end of the disease, and the blood is often greatly altered (see Hæmatemesis, Chapter XIII.).

In many cases the diagnosis of gastric catarrh is only possible at the time the patient is seen by means of a test-meal. The utilisation of this method not only shows the diminution in the amount of hydrochloric acid but the presence of mucus, and by it the degree of damage to the stomach can be ascertained—viz. whether in addition to the diminution in the secretion of hydrochloric acid, pepsin is present during digestion, as well as the curdling ferment. In intractable cases, such an investigation as this is of great importance not only for the purposes of diagnosis, but of treatment. In not a few cases of gastric catarrh, the diagnosis is evident from a consideration of the history, and from the characters of the vomited matters and of the symptoms, while others present a great difficulty in diagnosis unless the test-meal be resorted to. Epigastric tenderness is frequently absent, and in the later stages when there is degeneration, pain may be only slight, and no mucus may be present in the vomit. In these cases of atrophic stomach catarrh when first seen in this state, a test-meal remains the only means of diagnosing the condition of the stomach, although the previous history may point to the disease.

Treatment.—Gastric catarrh, being an inflammation of the stomach, although chronic in its course, must be treated like other inflammatory conditions. For an acute inflammation, rest is essential; for a chronic inflammation, rest is also necessary, and at a certain period of the disease stimulation. Rest can be given to the stomach by means of suitable food, food which the disordered organ can digest. It can also be given medicinally by means of drugs (Chapters X. and XI.). Although rest is one of the essentials of treatment, and important as allowing the chronic inflammatory condition to resolve, yet if food is to be taken into the organ and digested without discomfort, the disordered stomach must be helped. For in gastric catarrh, the inflammatory change leads to a great diminution in function of the organ; a diminished secretion of the gastric juice, especially of the hydrochloric acid, and a diminished motor power of the organ. As regards medicinal treatment, there is no remedy which may be said to be universally beneficial in gastric catarrh, except the mineral acids—nitro-hydrochloric and hydrochloric; these supply the deficiency of the gastric juice, and thus aid the process of digestion when given shortly after meals. For stimulation of the gastric secretion, alkalies before meals are necessary, with or without bitters. Not uncommonly, however, bitters irritate the stomach in gastric catarrh. As sedatives, morphine, opium, codeine, cocaine, and cannabis indica may be used (Chapters X. and XI.).

In chronic gastric catarrh, the regulation of the mode of living is of great importance; meals at regular intervals, not late in the evening, a moderate amount of exercise between meals, and a period of rest afterwards are essential. With subacute cases and in the exacerbations of chronic catarrh, bodily and mental rest is essential in the treatment, and, if not obtained, delay in recovery is increased. In chronic cases of catarrh, the supplementary methods of treatment (Chapter XII.) are of great use, viz. hygienic treatment, local or general massage, etc.

Speaking generally, it may be said that treatment in the early stages of gastric catarrh is very beneficial, as also in those cases where there are attacks at intervals. But in the later stages where there is degeneration of the mucous membrane, no permanent cure is possible, although much may be done to prevent irritation of the stomach by unsuitable food, and the patient may lead a comparatively healthy life although his gastric digestion is deficient.

Subacute Catarrh.—This condition has already been in part discussed; but it is of such great importance that a short separate account is necessary.

Acute gastritis is a rare affection of the stomach, subacute catarrh is by no means uncommon, and from the severity of its symptoms and its profound effect on nutrition, an appreciation of its pathology and of its symptoms is of great importance.

Its etiological factors are the same as those of chronic catarrh, and it may either be developed in the course of chronic catarrh as one of the acute or subacute exacerbations of the disease, or it may arise by itself, being unconnected with previous catarrh, being in some cases preceded by occasional periods of gastric irritation. The first mode of origin has been already fully discussed, the second mode is best illustrated by the case quoted below.

Subacute catarrh is characterised by great epigastric pain following the ingestion of food; a diffuse pain, which is only relieved by vomiting. Its origin is usually somewhat sudden, and epigastric pain may be the first symptom noted, followed in a week or more by vomiting. These symptoms increase, flatulence and nausea with profound loss of appetite and occasionally thirst appear, and the patient although at first partaking of an ordinary mixed diet, gradually reduces the amount of food, and leaves off the food accessories, until only milk and broth are taken, and even when the patient comes under observation this bland diet may cause distress and vomiting.

Dilatation of the stomach is a feature of subacute catarrh, which is never absent, the organ extending to the umbilicus or below it, and upwards into the left axillary region. There is, too, in many cases, great irritability of the stomach on manipulation. During palpation active contraction of the organ is induced which causes pain while reducing the size of the organ.

The complexion is pale, sallow, or even waxy-looking, and after the condition has lasted for some months, there is well-marked wasting. The bowels are usually constipated, and large quantities of mucus may be present in the stools.

In the stomach contents, either vomited or removed by the aspirator, there is always an excess of mucus and a diminished percentage of hydrochloric acid. In severe cases, no pepsin or hydrochloric acid appears to be secreted at all; in others active pepsin is found, but a very small proportion of free hydrochloric acid. Bacteria very readily grow in the liquid contained in the stomach, and are frequently found.

Course, Duration, and Prognosis.—The course is prolonged. It may be months before the patient recovers sufficiently to take the solid food of an ordinary diet without pain or vomiting. With proper treatment, however, these patients recover. The course is more prolonged in those of middle-age than in young adults.

Treatment.—The treatment is that of other acute gastric affections; the relief of constipation, rest in bed, and rest for the stomach either by an initial period of rectal feeding, followed by a bland diet, or by a bland diet at first in those cases where the irritability of the stomach is not extreme (Chapters X. and XI.).

As an illustration of the disease the following case may be quoted (see also Chapter V. p. 141, Case I.).

The patient, a female, aged forty-four years, by occupation a cook, and not addicted to the use of alchohol in any form, came to the hospital complaining of vomiting and pain in the stomach after eating solid food.

The *present illness* was of seven months' duration, and began with violent epigastric pain after eating solid food. This was followed in a month by vomiting after meals, and both symptoms have persisted and been severe, with flatulence and occasional acidity, up to the date of her application for advice. A little wasting had been observed, and the menstrual periods had ceased for four months.

She has had hard work, irregular meals, and has been accustomed to go long hours without food. She was subject to "dyspepsia" before she had a set of false teeth, but this was some time previous to her present illness.

The *examination of her present condition* showed a deficiency in general nutrition; she was pale and there was myotatic irritability of the chest muscles. The body temperature while under observation varied between 98° and 99° F., the pulse between 60 and 72, and the respirations between 20 and 28. There was no sign of disease in any part of the body other than in the stomach, and the urine contained no albumin or sugar, although it varied greatly in specific gravity (1006-1030), and showed an occasional excess of phosphates.

Digestive System.—There was almost complete loss of appetite, and thirst was well marked. The *tongue* was covered with a thick brownish-yellow fur, and was not flabby or tooth indented. The upper *teeth* were absent and replaced by a false set, the lower teeth were in a bad condition, decayed and broken. There was sharp intermittent pain in the upper part of the abdomen every five minutes, with the sensation of contraction and of a "ball in the stomach." This pain was increased by locomotion, by micturition, and by constipation. There was flatulence, and the thick fluid removed from the stomach on admission consisted of ropy mucus, with a small quantity of alkaline turbid liquid containing no pepsin and no peptones, but some coagulable albumin, and on microscopical examination showing numerous bacteria.

The bowels were greatly confined, and small stools were removed by enemata. They consisted of hard yellow fæcal matter and long strings of translucent, unpigmented mucus, enclosing a few particles of fæcal matter. On microscopical examination, the cylinders of mucus were seen to be laminated and to contain a few granular cells. There was no blood.

Physical Examination.—There were no abnormal signs in the chest. The liver and spleen were not enlarged, and there was no solid tumour in the abdomen.

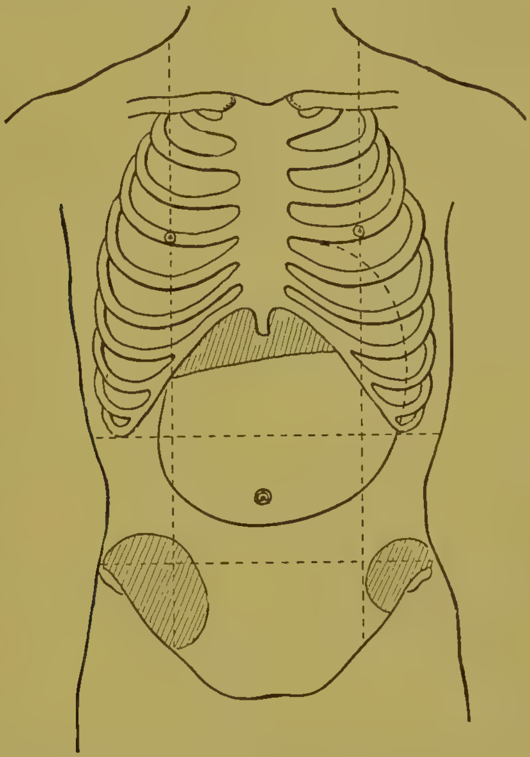

FIG. 30.—Diagram of the physical signs in subacute catarrh. Below the dilated stomach on the right was a prominence due to the distended cæcum, and on the left a similar one due to the distended sigmoid flexure.

The physical signs showed a dilated stomach, a distended cæcum and a distended sigmoid flexure, as seen in the accompanying diagram (Fig. 30).

The lower edge of the stomach could be felt as a rounded mass two inches below the umbilicus, and this edge could be traced to the left hypochondrium, but became ill-defined on the right side. The cæcum was detected as a rounded and tympanitic prominence in the right iliac region, and the sigmoid flexure showed a similar prominence on the left side. Manipulation of the stomach caused active peristalsis of the organ with the characteristic pain previously described, and there was diffuse tenderness all over the stomach region.

Course and Treatment.—The patient rapidly improved under treatment. She had complete rest in bed, and was fed solely per rectum (Chapter XI.) for thirteen days. Feeding by the mouth was then carefully begun. The irritability of the stomach disappeared; the organ diminished in size, and by the time the patient left the hospital she was taking a modified ordinary diet without discomfort.

Remarks.—In this case, which is a typical one, the etiological factors of the catarrh were: (1) a previous gastric irritation caused by improper mastication of the food; (2) the irregular mode of life—a life of hard work, irregular meals, and long hours without food; (3) the age of the patient. Alcohol was not a factor, as the patient was nearly a teetotaller.

The *diagnosis of the catarrh* rested on the following points:—

1. The relation of great and diffuse epigastric pain and vomiting to the ingestion of food.
2. The presence of mucus in the stomach and in the stools.
3. The absence of pepsin and hydrochloric acid from the stomach contents.
4. The dilatation and condition of irritability of the organ.

CHAPTER IX.

GASTRITIS TOXICA (TOXIC GASTRITIS)—GASTRITIS MYCOTICA (INFECTIVE GASTRITIS)—ATROPHY AND DEGENERATION OF THE STOMACH.

Two other forms of gastritis remain to be described: in one (toxic gastritis), the exciting causes are chemical poisons taken into the stomach; in the other (infective gastritis), the stomach wall is invaded by pathogenous bacteria, which enter either from the interior of stomach, or proceed from an infective focus in the body.

Toxic Gastritis.

A variety of poisons when swallowed act directly on the alimentary tract, and most violently on the parts with which they come into direct contact, viz. the mouth, pharynx, larynx, œsophagus, and stomach. As regards the œsophagus and stomach, their effects are important since when not immediately fatal, they, in many instances, cause considerable injury to the mucous membrane and lead to permanent disorder of digestion or to stricture.

These poisons may be classed as follows:—

1. Concentrated mineral acids: sulphuric, hydrochloric, nitric.
2. Caustic alkalies: chiefly potash.
3. Organic bodies: oxalic acid, carbolic acid, nitrobenzol.
4. Inorganic bodies: arsenious acid, corrosive sublimate, potassium chlorate, potassium cyanide, and phosphorus.

Of these poisons, phosphorus has an action differing somewhat from the rest, inasmuch as its corrosive action on the mucous membrane is not so evident. The mucous membrane may show congested areas, but the chief change is one discovered microscopically, and consists in a fatty degeneration not only of the epithelial lining but of the cells of the secretory glands; this change may extend to the submucous and to the muscular coat.

The other poisons mentioned act either as corrosives or as great irritants. Their primary effect is to destroy portions of the mucous membrane, the extent of destruction being dependent on the amount of poison taken, and the quantity of food in the stomach at the time. Mineral acids and caustic alkalies produce the greatest destruction; and the inflammation produced by mineral acids is much greater than that produced by oxalic acid, corrosive sublimate, carbolic acid, and arsenic. Around the areas of the mucous membrane actually destroyed by the corrosive, there is intense inflammation, which is shown by a swelling and reddening of the mucous membrane, and by the extravasation of blood to a greater or less extent, the blood rapidly becoming blackened. If the inflammation is not so great, an excessive secretion of mucus occurs, and the condition of the mucous membrane is similar to that already described in acute gastritis. Both in acute gastritis and in toxic gastritis there are irritants which produce the inflammation, but in the former case the irritants are not so powerful as in the latter.

Cases of toxic gastritis come under the charge of the physician in their early and acute stage and in the stage of recovery, when the acute inflammation has subsided, and there are anatomical changes in the mucous membrane, which are the cause of grave disorder of digestion.

1. In the parts most acted upon by the poison ulcers are found, superficial and deep. The locality in which they are found varies; sometimes at the cardiac orifice or in the central region of the stomach, sometimes in the pyloric region and at the pylorus. By healing and the formation of a contracting scar the organ is distorted, or when this occurs at the pylorus, obstruction is produced, leading to great dilatation of the organ (Chapter XV. p. 382, and Chapter XVI. p. 441).

2. In the parts of the mucous membrane not directly

affected by the concentrated poison, chronic inflammation is seen to a greater or less extent. These have been already described (Chapter VIII. p. 237). It is only necessary to repeat that there is congestion and an excessive secretion of mucus, leading eventually to an extensive fibrosis of the mucous membrane and even of the submucous and muscular coats, and to the degeneration of the secretory glands. The fibrosis frequently leads to a polypoid condition of the mucous membrane (*état mamelloné*), especially of the pyloric region, and even when there is no pyloric obstruction the organ may be dilated to a greater or less extent as well as cirrhosed. Small cysts are not infrequently found, and they may be extremely numerous.

These changes in the stomach are not infrequently associated with similar changes in parts of the œsophagus and small intestine.

Symptoms.—The symptoms referable to the poisonous effects of these toxic agents on the stomach come within the domain of toxicology. They may, however, be said to resemble those of acute gastritis, except that they are more pronounced; the pain is greater, the vomiting is more frequent and the retching violent, intensifying the pain. There is acute epigastric tenderness diffused over the stomach region, and there may be the signs of peritonitis. The appetite is lost, and intense thirst is frequently present. Symptoms of collapse are constantly observed and are shown by bodily weakness, a cold, clammy skin, and a small rapid pulse. Giddiness is a frequent symptom after the primary effect of the poison has passed off. Albuminuria and hæmaturia are not uncommon.

The characters of the vomited matters are of great importance. They may consist of the ordinary food contents of the stomach, with perhaps a great amount of mucus, or they may be strongly acid or alkaline, or contain more or less altered blood.

Strongly acid vomit may be due to poisoning by the mineral acids or oxalic acid: for the tests distinguishing this from cases of very acid vomit due to diseases, see Chapter V. Strongly alkaline vomit never occurs in disease; it is always a sign of poisoning. In the case of poisoning by arsenic, tartar-emetic, phosphorus, and carbolic acid, the reaction of the vomit may not differ from normal.

The presence of blood in the vomit may be due to poisoning by the mineral acids or the caustic alkalies, by arsenious acid, corrosive sublimate, and sometimes by phosphorus. With mineral acids, the blood is tarry; with the caustic alkalies, brown or black, and mixed with a very tenacious mucus; with arsenic, corrosive sublimate, and phosphorus, the blood may be red.

Besides the symptoms above enumerated there are others of a distinguishing quality, viz. a burning sensation in the mouth and throat, and in the chest, and great pain on swallowing liquids or solids. The stains of the poison may be found on the lips, tongue, cheeks, or fauces. With tartar-emetic, and especially with corrosive sublimate, there is a metallic taste in the mouth. There are laryngeal symptoms, suffocative cough and dyspnœa, in cases of poisoning by the mineral acids, oxalic acid, and the caustic alkalies. Diarrhœa is present in poisoning by arsenic, corrosive sublimate, tartar-emetic, and phosphorus, but is absent in poisoning by the mineral acids, carbolic acid, and oxalic acid.

Course and Duration.—When the patient does not die from the immediate effects of the poison, *i.e.* within a fortnight, the case then progresses as one of acute or subacute gastritis, the irritability of the organ with vomiting of mucus and epigastric pain being well marked. The subacute or acute symptoms may completely disappear and recovery take place, but inasmuch as the effect on the mucous membrane may have been primarily great, a permanent gastric disorder may be left. In mild cases this disorder takes the form of a chronic gastric catarrh with subacute exacerbations; in others, where there is much destruction of the mucous membrane, ulceration is produced, and there are the signs of gastric insufficiency with chronic congestion. The ulcer may be at the cardiac or pyloric end of the organ, and presents the same signs as the chronic ulcer of the stomach (Chapter XV.). It may heal and lead to stricture of the cardia or of the pylorus. The duration of such cases is prolonged and there may be no recovery, and the patient dies of inanition or of some intercurrent affection, pneumonia or tuberculosis.

Diagnosis.—Apart from its legal importance, the diagnosis of cases of poisoning in which the stomach is affected is of great importance to the practitioner, inasmuch as the life of the patient frequently depends on early recognition and early treatment. The practitioner may be called for the first time to see a patient with severe vomiting, perhaps of blood; and in such cases the absence of poisoning must be proved to his own satisfaction. In many cases there is no doubt of the diagnosis of poisoning from information supplied either by the patient himself or the friends, or by the presence of stains on the clothes worn or on the bed-clothes, or of stains and excoriations of the lips, mouth, and fauces. As a rule, from the characteristic signs already described as referable to the stomach, there is but little difficulty in the diagnosis of severe vomiting due to poisoning by the mineral acids, caustic alkalies, or by carbolic acid. Nor is there much difficulty in the diagnosis of most of the cases when they are acute; they originate suddenly, they occur after a meal or after something drunk, and a suspicion of the cause of the illness is soon settled by a chemical analysis of the egesta or the stools. But there are three classes of cases which are sometimes met with, and which require great care in diagnosis. These are cases of poisoning by foods, and chronic arsenical and antimonial poisoning.

Chronic antimonial poisoning is usually homicidal. The repeated administration of small doses of tartar-emetic produces the well-known effects of the drug, viz. nausea, vomiting (without hæmatemesis), diarrhœa, tenesmus and great prostration, which may end in death. The presence of diarrhœa and tenesmus distinguishes the condition from cases of subacute gastritis, in which there is constipation as a rule, and a chemical examination of the fæces or vomit decides the question.

Chronic arsenical poisoning is not infrequently observed. It may occur from continuous medicinal administration, but is more usually seen in those in whose occupation arsenic is handled, *e.g.* in arsenic factories, in the manufacture of wall-papers and of artificial flowers, and in chromo-printing. With the symptoms referable to the stomach, such as nausea, occasional vomiting, attacks of epigastric pain, and occasionally epigastric tenderness, there are other characteristic symptoms. Diarrhœa or the frequent passage of small motions not infrequently tinged with blood may occur, but is not continuous. There are the symptoms of coryza with a dry tongue and throat, associated with thirst; these are often the first signs. There is a characteristic eruption either on the face, the hands or the scrotum, and there may be a generalised eruption resembling eczema. One or other of these symptoms is rarely absent, and with the bodily prostration, the headache or the supervention of peripheral neuritis serves to distinguish the cases from that of any subacute or chronic gastric disorder.

In the homicidal cases of chronic antimonial and arsenical poisoning, the diagnosis of typhoid fever has been made, usually by the homicide himself. Such cases are readily distinguished from typhoid fever by the absence of the characteristic febrile rise of temperature, of the eruption and of enlargement of the spleen.

Poisoning by Foods.—It is impossible here to discuss all the aspects of poisoning by foods, but some notice of it is necessary, since the chief early symptoms are referable to the gastro-intestinal tract. The poisoning by food under consideration is that which occurs as the result of eating food, usually prepared food, which has undergone bacterial decomposition with the formation of chemical poisons. In other cases the poisoning appears to be due, not so much to the bacterial chemical products present in the food when eaten, but to the bacteria themselves, which develop in the gastro-intestinal tract and act as virulent poisons. Indeed, such cases may show an incubation period, and are really to be considered as a bacterial infection of the gastro-intestinal tract. Death may occur in both kinds of poisoning, and in the chemical form it may be rapid. By far the greater number of cases arise from pork, either pickled, or in the form of pie or ham. The next most numerous cases arise from tinned foods—salmon, sardines, and tongue.

The symptoms are those of irritant poisoning; there are epigastric and abdominal pains and cramp, followed by repeated vomiting or the passage of profuse watery stools. As a rule, there is no hæmatemesis or melæna. There is great prostration, the skin is cold and clammy, the pulse very frequent, small and compressible, and cramps of the limbs and muscular twitchings frequently occur. The course is usually afebrile, but there may be fever. The identification of the chemical poisons and of the bacteria in food which produce these symptoms is still very incomplete; but the symptoms clearly resemble those grouped as cholera nostras, choleraic diarrhœa, cholerine, etc. There are no doubt many different causes of the poisoning, both chemical and bacterial.

The diagnosis has to be made from acute arsenical and antimonial poisoning. The history often clearly points to a particular food eaten by a certain number of individuals, as the cause of the attack; an analysis of the food will exclude mineral poisoning, and an examination may lead to the separation of a poisonous chemical body or of a toxic bacterium. There is no hæmatemesis or melæna, as in poisoning by arsenic; and the motions are often extremely offensive, a point which will distinguish such poisoning from that by any acid, alkaline, or mineral poison.

Treatment.—The treatment of the acute symptoms in toxic gastritis does not come within the scope of this work. The results of the inflammatory condition are treated in the same manner as other chronic gastric affections; chronic ulcer, chronic gastric catarrh, and dilatation (Chapters X., XI., and XII.).

GASTRITIS MYCOTICA (INFECTIVE GASTRITIS).

The stomach is sometimes the seat of the specific lesions of certain infective disorders. Thus in diphtheria, the mucous membrane is in rare instances covered with a false membrane, and this may occur when diphtheria complicates scarlet fever and measles, as well as when it exists by itself. In smallpox an eruption similar to the pustules on the skin and larynx may be present on the mucosa; and tuberculous, typhoid, and syphilitic ulcers of the mucous membrane are in very rare instances found.

The term phlegmonous gastritis is applied to cases where either a localised abscess is formed in the stomach, or where there is a diffuse inflammation of the submucosa due to bacterial invasion, which usually ends in the formation of multiple foci of suppuration.

The majority of cases of phlegmonous gastritis which have been described occurred in the course of a general infection of the body, such as is present in puerperal and other forms of pyæmia, rarely in variola and in typhoid, or following wound infection.

A single large abscess may be formed which bursts into the stomach, the pus being vomited. More common than this is the form of diffuse infiltration of the stomach walls with pus. The process begins in the submucosa, and proceeds like all pus infection, in the formation of groups of leucocytes with liquid or fibrinous exudation. As the foci of suppuration extend they burst through the mucous membrane, emptying their contents into the stomach. The mucous membrane thus becomes riddled with sinuses, which are sometimes scattered throughout the whole of the organ, or are limited more particularly to the pyloric region. Some of the cases of multiple ulcers of the stomach occurring in pyæmia are formed in this manner. Not only the submucous coat but the muscularis and serosa may be infiltrated with pus, and the disease of the stomach is commonly associated with peritonitis.

The cause of the gastritis is in some instances a streptococcus (Ziegler), which is observed in numbers in and around the foci of suppuration; but the particular micro-organisms causing the inflammation have, in the majority of instances, not been sufficiently studied for identification. These cases are, indeed, far from common.

E. Fraenkel[1] has described a case of diffuse and acute inflammation of the submucosa with effusion of blood, and the formation of bladders of air; around the foci of inflammation numerous bacilli of one form were discovered. The gastritis occurred in a healthy man of thirty-five years of age, who had sustained a compound fracture of the right index finger. An attempt was made to save the finger, which apparently did well, without showing any signs of bacterial infection. Seven days after the injury, symptoms of acute gastritis supervened, in slight fever, great epigastric pain, bloody vomiting, and collapse. Slight jaundice appeared and the patient died in two and a half days after the onset of the symptoms. The explanation of the case appears to be that the bacteria found in the stomach walls were presumably the cause of the gastritis, and were derived from the wound; but the case is by no means clear. The rest of the body showed no great change, and there was no general bacterial infection of the organs.

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[1] "Ueber einen Fall von Gastritis acuta emphysematosa, wahrscheinlich mykotischen Ursprungs," *Virchow's Archiv*, 1889, Bd. cxviii. p. 526.

Most of the cases of infective gastritis are, as has been said, secondary to some infective process going on in the rest of the body. Deininger[1] has published a case which was apparently primary. The gastritis was extreme and was of the diffuse purulent form which has been already described, and there was no other pus formation in any other part of the body. It occurred in a drunkard, aged fifty-three years, with well marked cirrhosis of the liver, as well as chronic catarrh of the stomach and intestinal tract. In cases of cirrhosis of the liver, bacterial infection of the body may be the immediate cause of death, as I have myself observed; and in Deininger's case the presence of chronic catarrh of the stomach might be considered the local predisposing cause for the invasion of the pus-forming micro-organisms from without.

Klebs[2] has described two cases of the disease under the title of *Gastritis bacillaris.* In one, there were present in the mucous membrane, chiefly along the greater curve, rows of large and small (five to ten millimeters in diameter) brownish areas, some becoming confluent. Where these spots were present, the mucous membrane was thickened and hard, and showed on microscopical examination a collection of leucocytes and numerous bacilli free on the surface of the membrane and in the lumen of the glands. The bacilli extended throughout the thickness of the stomach wall; a few were found in the kidneys. There was slight jaundice, as well as hæmorrhagic nephritis. The second case was similar, but erosions of the mucous membrane were present, and there was advanced red atrophy of the liver.[1]

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[1] "Zwei Fälle von idiopathischer Gastritis phlegmonosa," *Deutsches Arch. f. klin. Med.*, Leipzig, 1879, Bd. xxiii. p. 624.

[2] "Allgemeine Path.," 1887, Bd. i. p. 206.

In anthrax occurring in man, the stomach may be the seat of invasion of the specific bacillus. I have seen this in one case[2] that occurred at Guy's Hospital, in which the primary seat of infection was in the left cheek where a malignant pustule developed. After death, besides the usual appearances presented in cases of anthrax, the anterior wall of the stomach was swollen and infiltrated throughout with pale, slightly turbid serum. Large numbers of anthrax bacilli were discovered in sections of the stomach wall.

The majority of cases of infective gastritis are chiefly of pathological interest. Recovery may take place, and in that case scars are formed in the position of the abscesses[3] (Diettrich).

ATROPHY AND DEGENERATION OF THE STOMACH.

The chronic inflammatory changes which have been considered under the heading of catarrh produce atrophy of the stomach walls and chiefly of the mucous membrane; since the process, whether it leads to a general thickening of the walls or of the mucous membrane, or in rare cases to a thinning, ends in a destruction of the secretory gland tissue. There is, however, another form of atrophy which is sometimes found, and which is not, so far as is known, associated with inflammatory changes, viz. a primary atrophy. Pathologically and, indeed, clinically, this form of atrophy is quite distinct from that occurring as the result of inflammation. Its cause is not quite clear, and it is usually discovered post-mortem without producing during life any definite symptoms pointing to advanced disease of the stomach. Such patients are old and feeble, can take but little food, and that not of an irritating kind, and so there are no symptoms of irritation of the organ; while the food that is taken is readily digested in the duodenum. What symptoms such a condition produces are masked by the general condition of the patient. With some cases of pernicious anæmia, also, atrophy of the stomach is associated.

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[1] A similar case was published by Nasse, in which hæmatemesis occurred from erosion of the left superior coronary artery.

[2] *Journal of Pathology and Bacteriology*, vol. i.

[3] For further information on Phlegmonous Gastritis, see Leube, "Ziemssen's Handbuch der speciellen Path. u. Therap.," vol. vii., Hft. 2, 1879. The literature up to that date is given—in all of 31 cases, 22 of which were collected by Auvray, *Thèse*, Paris, 1866.

Non-inflammatory degeneration of the stomach may be divided into three classes:—

1. Primary atrophy.
2. Fatty degeneration of the glands occurring in cancer of the stomach or of some other part of the body, and in some cases of long-standing ulcer.
3. Albuminoid degeneration.

Primary atrophy of the stomach is not a very common disease, and occurs chiefly in patients beyond middle age, as in the similar condition occurring in other organs, for example bone, the uterus and the kidneys. The ages of Fenwick's[1] cases varied between 43 and 75; in Meyer's four patients the age was over 60 years (64 to 78); in Rosenheim's two patients, 36 and 61 years; in Lewy's and Ewald's patients, 70 and 67 years respectively; in Schirren's patients, 29, 33, and 52 years; in Litten's and Einhorn's patients, 19 and 21 years. It is probable that in some of these cases the condition of primary atrophy was mistaken for the degeneration following inflammation. In atrophy the stomach walls are greatly thinned, in some cases so as to be semi-transparent; the normal appearances of the whitish-yellow opaque mucous membrane, arranged in rugæ, being lost. The organ is dilated and more or less empty, and there is no post-mortem digestion of the mucous membrane. The absence of post-mortem digestion, as well as the fact that no pepsin or hydrochloric acid is secreted by such a stomach, shows that the secretory glands of the organ have completely lost their function.

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[1] "On Atrophy of the Stomach," by S. Fenwick, London, 1880. For further literature on Atrophy of the Stomach, see Handfield Jones, *Trans. Path. Soc.*, London, vols. iv. and v.; *Med.-Chir. Trans.*, London, 1854; G. Meyer, "Zur Kenntniss der sogenannten Magen-atrophie," *Ztschr. f. klin. Med.*, Berlin, 1889, Bd. xvi., p. 366; Litten, *ibid.* vol. xiv. p. 573; Rosenheim, *Berl. klin. Wchnschr.*, 1888, Nos. 51 and 52; Lewy, *ibid.*, 1887, No. 4; Ewald, *ibid.*, 1886, No. 32; Schirren, "Inaug. Diss.," *Kiel*, 1888 (quoted by Meyer).

Microscopically, the glands are found in an advanced stage of degeneration. In many parts no remains of the glands can be seen at all, or only indications after the tissue is treated with acetic acid. Where the glands are absent connective tissue only is seen, with fatty granules. In other parts the shape of the glands can be distinguished, but instead of being lined by epithelium they are filled with fat globules and detritus, the cells having undergone complete fatty degeneration. The submucous tissue and the muscular coat also waste.

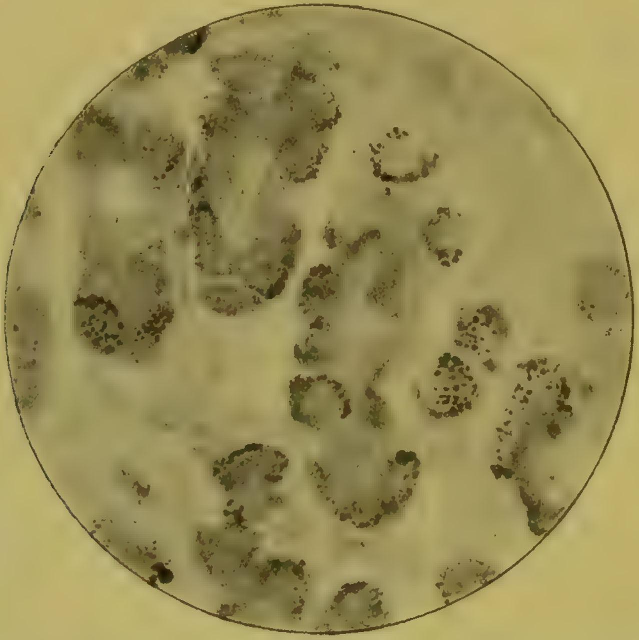

FIG. 31.—Fatty degeneration of the cardiac glands in a case of gastric ulcer without catarrh. From a photograph, × 150. The outlines of the glands are seen, and in many parts there are fatty granules stained black by osmic acid. From a preparation hardened in osmic acid, but not counterstained.

In some cases of pernicious anæmia, atrophy of the stomach occurs, and where death has been ascribed to atrophy of the stomach, sufficient attention has not in all the observations been paid to the condition of the blood with the view of excluding the presence of pernicious anæmia.

Fenwick has shown that in cases of cancer of the pylorus, and in cases of cancer elsewhere (mamma and uterus especially), the gastric glands have sometimes been found in a state of fatty degeneration; they may be completely destroyed, their place being taken by fibrous tissue. This fatty degeneration (non-inflammatory) of the gastric glands in cancer does not stand alone in that disease. Fatty degeneration of the heart muscle, of the liver, and of the cortex of the kidney constantly occurs. It is a degenerative change in part due to the anæmic condition of the patient (see Cancer of the Stomach, Chapter XVI.).

In cases of long-standing ulcer of the stomach, fatty degeneration of the glands may be found, accounting in part for the deficient digestion which is present in these cases. Fig. 31 represents the form of degeneration that occurs. The cells of the acini are seen to contain fat granules which stain deeply with osmic acid.

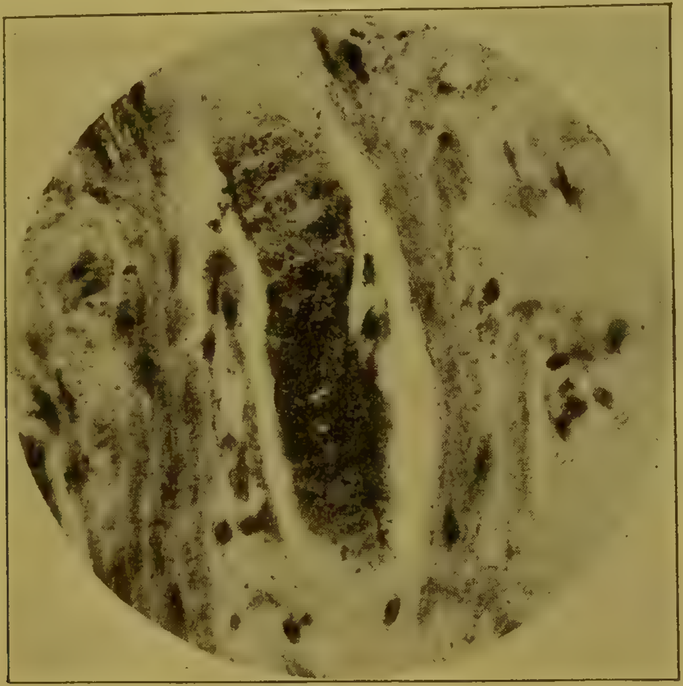

FIG. 32.—Albuminoid degeneration of the gastric mucous membrane. From a photograph, × 350. From a case of advanced pulmonary tuberculosis. The figure shows a portion of one gastric gland, in which the cells have become fused together, and have, for the most part, lost their nuclei. Fatty granules are also seen in the cells. On each side of the gland, unstriped muscle fibres are seen which have undergone albuminoid degeneration. The cells are swollen and have become structureless, the elongated nuclei being in some parts completely absent. From a preparation hardened in Marchi's fluid, and stained with logwood.

Albuminoid degeneration of the stomach occurs chiefly in long-standing cases of pulmonary tuberculosis and of syphilis, and in cases of prolonged suppuration. It affects chiefly the mucous membrane, but the muscularis may also show areas of degeneration. In the mucous membrane, not only are the vessels affected, but also the muscular fibres and the connective tissue between the glands. Fig. 32 shows the effect on the glands themselves; the cells are in parts homogeneous and in other parts fatty, while the nuclei have for the most part disappeared.

Symptoms.—These may be very few, and may be masked by the presence of a more serious disease, viz. pernicious anæmia or cancer. Otherwise such cases are associated with the symptoms of gastric insufficiency (*q.v.*), the anæmia and the emaciation being especially well marked.

Treatment is that of permanent gastric insufficiency (Chapter XI.).

CHAPTER X.

TREATMENT OF ACUTE AND CHRONIC AFFECTIONS OF THE STOMACH.

Medicinal Treatment.

The treatment of stomach affections has to be directed partly to the relief of the disordered or diseased condition, and partly to the relief of individual symptoms. "Specific" remedies, whether medicinal, dietetic, or hydropathic, do not exist for the treatment of diseases of the stomach. The successful treatment of functional disorder of the stomach differs in some respects from that of many other local disorders, in the fact that although a particular treatment may be used and be beneficial, yet its successful issue depends as largely on the patient as on the practitioner. For in most instances, the treatment interferes, often seriously, with what to many people is of prime importance, indulgence in favourite and usually harmful articles of diet. The patient who suffers acutely or moderately from a stomach affection readily submits to treatment till he considers himself better; he then is very apt to lose caution, and a relapse occurs after a period of intermission of his disorder. In this respect the practitioner can be of great aid to the patient by impressing upon him care in the regulation of his life and of his diet, so that his work is rendered more useful, his recreation more pleasurable, and undoubtedly his life is prolonged.

It will have been made evident from the foregoing chapters that the disordered or diseased conditions of the stomach which require treatment are the following:—

1. Alterations in the secretion of the gastric juice; hyper-

acidity due to hydrochloric acid, deficient acidity due to a diminution in the secretion of hydrochloric acid. In some cases diminution or absence of pepsin.

2. Alterations in the motor activity of the stomach, shown chiefly in diminished motor power, sometimes by irritability of the organ. Diminished motor power may end in dilatation of the organ.

Conditions of absorption do not admit of direct treatment; but as the interference with one function of the stomach disorders the remainder, the treatment of defective absorption becomes really part of the treatment of disordered secretion and disordered motility.

3. With these primary changes may be associated other conditions which require treatment.

a. Dilatation of the organ, whether simple or obstructive.
β. Bacterial fermentation of the food.
γ. Congestion of the organ; whether mechanical or active.
δ. A nervous irritability, set up by various local and general conditions, such as hyperacidity, irritant food (alcohol), or by ordinary food in certain conditions of the nervous system and in congestion of the stomach.
ε. Lastly, organic disease of the organ; ulcer or carcinoma.

It may be a matter of urgency to treat one particular symptom — pain, vomiting, flatulence, acidity—before treatment of the other defects in the functions is attempted; and, although organic diseases of the organ (ulcer and cancer) require special treatment, yet they are commonly accompanied by one or other of the disordered conditions enumerated. Whether primary or secondary, it is the disorder of the stomach functions which has to be treated, in order (1) if possible, to restore the functions to their normal activity, or (2) if this is not possible, to enable the organ to do as much work as it can.

In the treatment of stomach affections, therapeutic measures are adopted:—

1. To stimulate the stomach functions; secretion and motor activity.

2. To act as sedatives to an increased functional activity, *e.g.* an increased secretion of HCl, an increased motor activity.

3. To act as sedatives to nervous and motor irritability.

4. In some cases to supply a deficiency of secretion; hydrochloric acid and pepsin.

5. To prevent and counteract bacterial fermentation of the food.

6. To treat prominent symptoms, such as flatulence, acidity, and dilatation of the organ.

The therapeutic measures fall into two classes, general and local.

The general measures are those directed to the improvement of the general health of the individual; such as the regulation of the mode of living, general personal hygienic treatment, the administration of remedies for improving the general health, and the adoption of measures with the same object (massage and baths, Chapter XII.). In individual cases, general treatment forms the chief part of the therapeutics of stomach disorder; but in the majority of cases local treatment is essential.

Local treatment is partly medicinal, partly dietetic, and partly a resort to the mechanical methods of washing out the stomach and forced feeding, and to the methods adapted to the strengthening of the motor activity of the organ (such as local massage and the application of electricity).

Before proceeding to deal in detail with the therapeutic measures, it is necessary to recapitulate what has been said as to the essential characteristics of the stomach conditions, to which the foregoing remarks apply—viz. gastric irritation, gastric insufficiency, and chronic gastric catarrh. Each of these may exist by itself or be present with organic disease of the stomach, ulcer or cancer. They, moreover, may present certain symptoms in common, so that their treatment may in certain cases be the same.

It was shown that in gastric irritation, the tendency of the disorder was to a hypersecretion of hydrochloric acid, associated with irritant symptoms of the organ due chiefly to the character of the food taken. In gastric insufficiency, the condition was one of deficiency of the functions of the organ, produced by some general disease or by some unhygienic condition. In gastric catarrh, there is a deficiency of functional activity of the organ; shown in the first instance by a great diminution in the amount of hydrochloric acid secreted, later on by a diminution of the pepsin. To this is added congestion, which is shown by the irritant symptoms, more marked than in gastric irritation.

In all these conditions there may exist a condition of nervous irritability of the stomach, as shown by repeated eructations, vomiting and retching, pain, direct or referred. In gastric irritation this is produced by the continued irritation of the organ; in "nervous" dyspepsia and in gastric insufficiency it is due to a condition of the general nervous system primarily, whether produced by a blood condition or by a functional nerve disorder; in gastric catarrh it is induced by the chronic congestion of the mucous membrane and by the irritation of food. In gastric insufficiency and chronic gastric catarrh, the motor activity of the organ is sooner and more profoundly affected than in gastric irritation, and there is frequently found a persistent moderate dilatation of the organ, associated in some cases with bacterial fermentation.

In all these conditions there is a tendency to delay of food in the organ, from the various causes already fully described, and the continued delay of food further aggravates the disordered condition.

The treatment of affections of the stomach will be discussed under the following headings:—

1. Medicinal treatment.
2. Dietetic treatment (Chapter XI.).
3. Washing out the stomach; massage and hydrotherapeutic and other treatment (Chapter XII.).

Medicinal Treatment.

By medicinal treatment, not only may the functions of the stomach be affected (secretion, motor activity, and thus absorption), but excessive acidity due to hydrochloric acid or to organic acids controlled, pain and irritability relieved, flatulence and fermentation prevented.

It was shown previously (Chapter I.) that the usual secretion of the gastric juice readily takes place in response to mechanical, thermal, electrical or chemical stimuli; but that the only stimulus which produced a prolonged secretion was the presence of digestible food in the stomach. Of chemical stimuli, the most important are dilute alkalies, common salt, and alcohol.

A consideration of the process of normal digestion shows that it is probable that the continued secretion of gastric juice during the period of digestion is dependent on the effect on the food of the gastric juice first secreted. Not only do the stomach contents become acid, but products (peptones) are formed which may after absorption stimulate secretion (Schiff). Sodium chloride is taken with the food, and thus stimulates secretion. In the healthy stomach the secretion of hydrochloric acid varies from time to time. Thus in the early period of digestion, its place may be taken by organic acids (chiefly lactic acid), liberated from the food: after this is absorbed, the secretion of hydrochloric acid begins. Again, if from any cause the contents of the stomach become too acid, the secretion of hydrochloric acid ceases or diminishes; so that the amount of free acid in the stomach contents has an appreciable effect on the continuous secretion of hydrochloric acid. In the normal digestion of food containing a large quantity of proteids, the hydrochloric acid combines with these proteids, and in this condition, although the proteids can still be readily digested by the pepsin, yet the acid combined with it cannot be considered as free acid. The free acid in the stomach is thus continually diminishing, and a renewed quantity of hydrochloric acid is being secreted. The dependence of the secretion of hydrocloric acid on the amount of free acid in the stomach contents cannot be too much insisted upon in a consideration of the medicinal treatment of stomach disorders.

Moreover, the continued movements of the stomach, although dependent on a nervous mechanism, are closely related to the process of digestion, which acts as a stimulus and a continuous one. Many agents—mechanical, thermal, electrical, chemical—act as a local stimulus to the movements, but the effect is only momentary; the stimulus of digesting food acts as a continuous stimulus. This result can only occur from a gentle and varying stimulation, and there can be but little doubt that the varying amount of the secretion of the hydrochloric acid acts as one of the main stimuli of this continued movement. The stimulus of food as a mere mechanical agent is greatest during the early period of digestion, while as a chemical agent it is greatest at the time of highest acidity of the stomach contents. Absorption appears to be intimately related to the movements of the stomach and to the condition of the blood supply; and, indeed, running through all the previous chapters is the theme that the functions of the stomach are mutually dependent, insomuch that when one is disordered the others are also affected.

With these general considerations in mind, the subject of the action of drugs on the stomach in disease may be discussed.

1. *Secretion and Digestion.*—Secretion in disease is chiefly influenced by acids, alkalies, and alcohol. The effect of alcohol in stimulating secretion has already been fully discussed (p. 53).

Alkalies.—Alkalies in small doses given in watery solution before meals stimulate the secretion of gastric juice; this has been proved experimentally. In disease, as in health, they have this action; but their action in disease is not a simple stimulation of secretion. As has been shown the stomach in many cases of gastric irritation is not empty before the next meal is taken; it contains perhaps only a small quantity, but still an appreciable quantity, of an acid liquid which, when neutralised by an alkali, is readily expelled from the stomach. Similarly in cases of bacterial fermentation, without great dilatation, this action is also seen; not only does the alkali stimulate the secretion, but it prepares the stomach for the next meal by neutralising the acid liquid it contains.

Alkalies after meals cannot be said to have much effect on secretion, but they have an important effect on digestion. Thus, if alkalies are given after meals when there is no hyperacidity of the stomach contents, digestion is delayed often to a marked extent owing to the neutralisation of the free acidity. If, on the other hand, there is hyperacidity, this is reduced by the alkali given and the digestive process is hastened. It is not simply that the diminution of hyperacidity increases the chemical process of digestion, but that thereby the movements of the organ are made more regular, because there is perhaps no condition of the stomach contents which interferes so much with the regular movements of the stomach as hyperacidity. The action of alkalies in stimulating secretion when taken before meals is only a temporary one; it can only be considered as a starting of the secretion, which in the absence of the stimulus of food soon ceases. The only chemical substance which, as far as we know, causes a continuous secretion of the gastric juice, is alcohol, and this prolonged secretion may go on after food has left the organ. This effect of alcohol is of very great importance, not only in the etiology, but in the treatment of gastric affections. It is, however, in but few cases, and those chiefly of gastric insufficiency, that this effect of alcohol is beneficial; in gastric irritation, and in most cases of catarrh, alcohol is harmful.

One important effect therefore of alkalies is to act as *antacids,* and with this object they are given sometimes before meals in the conditions mentioned above, but usually after meals to diminish the hyperacidity of the stomach contents, whether due to increase of hydrochloric acid or to the formation of organic acids in bacterial fermentation.

The following alkalies are used as stimulants to secretion or as antacids:—

	As stomachic before meals.	As antacid between meals.
Liquor potassæ . . .	5–10 minims.	15–30 minims.
Potassium bicarbonate . .	10–20 grains.	20–30 grains.
Sodium bicarbonate . .	10–20 grains.	30–40 grains.
Ammonium carbonate . .	5–10 grains.	
Creta præparata . . .	Not used.	5–10 grains.
Lime water	Not used.	½–1 ounce.
Saccharated lime solution .	Not used.	15–60 minims.
Magnesia	Not used.	10–60 grains.
Magnesium carbonate . .	Not used.	5–10 grains.

A useful antacid powder is that recommended by Trousseau[1] consisting of—

Bicarbonate of sodium . . .	5 grains.
Creta præparata	10 grains.
Magnesium carbonate . . .	5 grains.

One powder to be taken after each principal meal and before going to bed.

As regards the other alkalies mentioned above, the most suitable for continuous administration is sodium bicarbonate; potassium carbonate, after a time, causes great bodily depression. In cases of functional disorder of the stomach in bronchitis these alkalies may be combined with ammonium carbonate in the mixture.

Great care must be exercised in the administration of alkalies as stimulants to secretion in cases of gastric insufficiency, inasmuch as they interfere with digestion if given in too large doses. It is best to begin with a dose of not more than 10 grains of bicarbonate of sodium before meals.

Acids, when given before meals, diminish to some extent the secretion of gastric juice. The effect can only be a temporary one, since the acid rapidly disappears from the empty stomach. Given after meals, acids temporarily increase the acidity of the stomach contents, and thus to some extent increase the rapidity of the process of digestion. The increase of free acidity is, however, undoubtedly very slight, since the acid soon combines with the proteids present. In disease acids are given after meals, as an aid to digestion. In hyperacidity of the stomach contents, however, they are harmful because of the increase of acidity they cause, and they often in these cases produce an aggravation of the symptoms. (For other actions of acids, see pp. 295, 305.)

Chloride of sodium is an important adjunct to the food inasmuch as not only does it directly stimulate the secretion of the gastric juice, but it provides the material for the secretion of the hydrochloric acid. In "chlorine hunger" the hydrochloric acid present in the stomach diminishes and may cease. In bottle-fed infants the useful action of chloride of sodium is more evident than in adults; in diluting the milk, the proportion of the salt is diminished, and if enough salt is not added to repair the deficiency, the infant frequently shows signs of impaired nutrition. In adults common salt is present in the solid food and is used in cooking.

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[1] "Lectures on Clin. Med.," *New Syd. Socy.'s Trans.*, vol. iii. p. 537.

Other drugs taken before meals, or soon after meals, increase the secretion of the gastric juice. Both quinine and iron salts have this action, especially where there is a functional diminution of secretion as in gastric insufficiency. Bitters also, no doubt, have a similar action, although experimentally they have been shown to have no direct stimulating action on the stomach; they may act indirectly by stimulating a flow of alkaline saliva, and may increase the appetite by gently irritating the stomach.

2. *Motor Activity.*—The treatment of a diminished motor activity of the stomach does not depend solely or even chiefly on medicinal agents. These, however, are useful in many cases; some have a direct action, others are indirect. As examples of direct stimulants to the motor activity of the stomach, strychnine and nux vomica, alcohol, and creasote may be cited; and part of their beneficial effects in gastric disorders is to be ascribed to this action. Alkalies are both direct and indirect stimulants to motor activity. When the stomach is empty they act as direct stimulants; if the stomach contains acid digesting food, they are soon neutralised and thus cannot act in this way. It is in cases of hyperacidity that their indirect action on motor activity is most marked. Thus in hyperacidity occurring after meals, as in gastric irritation, the tendency is for the movements of the organ to diminish. Whether this is due to the mere presence of very acid contents, or to this in association with a large excess of food, is not clear, but it is certain that when this hyperacidity is relieved by the administration of alkalies that the movements become more vigorous. The movements of the stomach in such cases appear, indeed, to be partly inhibited by its hyperacid contents. The administration of alkalies causes an ebullition of gas, which undoubtedly acts as a stimulant to the movements (partly by distention of the organ, partly by an action like that of a stirring rod), and the contents of the stomach are sometimes wholly, sometimes in part, expelled into the duodenum. A

similar action is seen on giving alkalies before meals when the stomach contains the hyperacid residue of the previous meal.

Antispasmodic remedies come under the heading of drugs that act on the motor activity of the stomach. The organ contracts spasmodically chiefly in two conditions: when a meal containing very irritating articles of food has been taken, and when the organ is full of gas which it cannot expel. Antispasmodics taken in these conditions relieve the spasm, chiefly by acting as a sudden stimulant to the motor activity of the organ by which the contents are expelled. Thus in the case of distention by gas, this is expelled both by the mouth and into the duodenum. In other cases, the spasm is relaxed and the movements of the organ recommence. The explanation of the relaxation of the spasm is not forthcoming, but it may be said that relaxation of spasm by drugs may be due either to slight and continued stimulation, to a passing paralysis of the muscle, or to the sudden stimulation already mentioned, whereby the offending material is got rid off.

The antispasmodics which are useful in stomach affections are:—

Spiritus chloroformi in doses of 10–15 minims. Spiritus etheris in 15–30 minim doses.
Ammonia, as in spiritus ammoniæ aromaticus (15–30 minims).
Creasote (1 minim doses) and carbolic acid, as glycerinum acidi carbolici (5–10 minims).
Cajeput oil, 1–3 minims on sugar.
Capsicum, 1–3 minims of the tincture.
Asafœtida, as tincture (½–1 fl. dr.) or in pill, and valerian as tinctura valerianæ ammoniata (½–1½ fl. dr.).
Hot water, mustard, pepper, peppermint, and ginger.

3. *Absorption.* — The promotion of absorption by the stomach cannot be accomplished by drugs. The treatment of motor inactivity of the organ has a great effect on absorption, as has been said, and as the stomach functions are restored to activity, so does absorption improve.

In one respect, absorption may be said to be treated in disease, and this is when a layer of tenacious mucus lines the stomach. Alkalies either in medicinal doses or when used for washing out the organ will help to remove this and so promote absorption.

Bitters.—It is extremely difficult to explain the action of bitters in gastric disorders. Experiment seems to show that they have no appreciable effect either on the secretion or on the movements of the organ in healthy animals. They may, however, have an action on the disordered stomach, but this it is impossible to define. One action they possess is certain, viz. that they increase the flow of saliva, which being alkaline may excite the secretion of gastric juice when swallowed. They appear to excite some slight secretion of gastric juice, but not to a greater extent than other mild irritants, and not greater, according to some, than distilled water. In chronic stomach disorders they are usually tolerated, but in some cases they act as irritants, and some, *e.g.* gentian, may produce diarrhœa. Their use is best limited to chronic functional disorders or to chronic gastric catarrh, and in all cases of acute stomach disease they are best withheld. They are best given as infusions in mixtures with alkalies or acids. The most serviceable bitters are compound infusion of gentian, infusion of quassia and infusion of calumba. Condurango is also not infrequently useful; it may be given in the form of liquid extract in doses of 10 to 60 minims, or of vinum, in doses of half an ounce to an ounce.

4. *Remedies Supplying a Deficiency of Secretion.*—These are free mineral acids and ferments — pepsin, pancreatin, papain.

Acids.—The chief diseased conditions of the stomach in which the hydrochloric acid is deficient are gastric insufficiency, gastric catarrh, atrophied conditions of the mucous membrane, and cancer. In the later stages of some cases of ulcer it may also be greatly deficient. Acids given with meals or soon after meals in these conditions actually supply a deficiency which is a necessity for proper digestion. Gastric digestion proceeds most rapidly in the presence of free hydrochloric acid, to a less extent in the presence of nitric and sulphuric acids. In the treatment of disordered digestion either the acidum hydrochloricum dilutum (containing 10·58 per cent of HCl) or the acidum nitro-hydrochloricum dilutum (the acidity of which is $\frac{1}{10}$ higher than that of the dilute hydrochloric acid) may be given. Both are administered in small doses, from 7 to 15 minims and if the meal taken is even as small in bulk as half a pint, this proportion of acid is very small; it will be diluted indeed to about 0·03 per cent HCl. There are, however, reasons explaining the useful action on digestion of even this small quantity of acid. In the first place, in the disordered conditions in which acids are given there is deficient absorption; this is probably true for gastric insufficiency as well as for gastric catarrh, hence the acid administered does not soon disappear from the stomach. Again the acid, as has frequently been explained, combines with the proteids present, and in this acid condition the proteids are readily acted upon by pepsin. The combination of acid with proteids, moreover, readily takes place in such cases, as the patients can only take a simple diet containing a large proportion of the proteids in an uncoagulated state. It may also in some cases be that the acid transforms the pepsinogen into pepsin, and thus more ferment is present than when acids are not given (p. 102). Whatever may be the exact explanation of the action of acids in these conditions, there is no question that they are highly beneficial. It is sometimes said that they act as gastric "tonics," but this is no explanation, and some such explanation as that given is probably near the truth.

Ferments.—The value of the administration of ferments in gastric disorders is not easy to determine. In the first place, many of the preparations which are sold as pepsin and pancreatin are almost, if not quite, inert as proteolytic ferments. The preparation of a ferment in an active state is a delicate operation and is often only imperfectly understood. Moreover, the activity of these preparations greatly diminishes in the course of time, so that all old preparations ought to be rejected. Of the preparations now obtainable the best are those in powder; a ferment kept in a perfectly dry condition retains its activity much longer than in solution.[1] Pepsin digests proteids only in an acid medium, and best in one containing hydrochloric acid to the amount of 0·2 per cent. Pancreatin (trypsin) digests proteids slightly in a neutral medium, but energetically in an alkaline one, *i.e.* one containing sodium carbonate to the amount of $\frac{1}{2}$ to 1 per cent. Papain is closely allied in its action to trypsin, inasmuch as it acts best in an alkaline medium. Pepsin as sold in powder is either the dried scrapings of the mucous membrane of the pig, which gives a very active ferment but contains a large amount of insoluble matter, swelling up in water; or a filtered extract of the mucous membrane, dried at a low temperature. This latter preparation is often only feebly active; it is, however, possible to obtain it in an active state and it is better than the "insoluble" pepsin for prescribing. Pancreatin is either the dried scrapings of the pancreas or the purified dried extract; and it is prepared in a liquid form as "liquor pancreaticus." Owing, however, to the fact that great precautions are necessary in preparing an active ferment many of the preparations in commerce have but little tryptic (proteolytic) action, although they may contain an active diastatic (amylolytic) ferment. Hence there is great necessity for testing the activity of both pepsin and pancreatin preparations before prescribing them. Papain is the dried purified extract of the green fruit of the Papaw tree, *Carica Papaya.* It is a proteolytic ferment, but its activity has been greatly exaggerated.

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[1] When pepsin or pancreatin are prescribed it is best either to test the activity oneself or to get this done or guaranteed by a skilled pharmaceutical chemist.

The Conditions of Administration of Ferments in Gastric Disorder.—Ferments are administered in functional disorders of the stomach to supply a deficiency in the amount of pepsin secreted. As a rule they are administered very indiscriminately, and the reports as to their valuable effects are on the whole illusory. In discussing the pathology of functional disorders of the stomach, it was shown that when secretion was diminished, the amount of hydrochloric acid was first affected, and that later on in the disorder pepsin might diminish as well, or even disappear. In some cases the disappearance of pepsin is accompanied by the secretion of inactive pepsinogen, which requires only free acid to change it into active pepsin. The practical point is, therefore, that pepsin is never diminished unless the secretion of hydrochloric acid is also affected, and as digestion is only possible in the presence of this acid, it is useless to give pepsin alone to supply a deficiency of secretion; it must be given with hydrochloric or nitro-hydrochloric acid.

The conditions in which pepsin (as well as hydrochloric acid) is diminished or absent are:—

1. Gastric insufficiency.
2. Gastric catarrh in the acute stage or in the later stages of the chronic.
3. In atrophy of the mucous membrane, whether primary or following catarrh.
4. In the later stages of chronic ulcer, due to prolonged gastric insufficiency or to catarrh.
5. In carcinoma ventriculi, due to the invasion of the mucous membrane by the new growth, to degeneration of the glands, or to the effects of catarrh on the mucous membrane.
6. In cases of prolonged dilatation of the stomach from whatever cause.

It is by no means advisable in all the cases enumerated to give pepsin in order to supply the deficiency in the secretion. Its administration must be guided by the consideration as to whether a recovery of the stomach functions is possible. In such cases, although it may be necessary to administer acids, pepsin is unnecessary, inasmuch as it is not deficient to so great an extent as hydrochloric acid, and its administration would tend to delay recovery of the gastric functions. In such cases as those of gastric insufficiency not due to any serious disease, such as pulmonary tuberculosis and chronic Bright's disease, by improving the general health and by local treatment the stomach soon resumes its ordinary functions. When there is, however, a serious disease, such as those mentioned or any of the others discussed under the heading of gastric insufficiency, the administration of pepsin and hydrochloric acid may be advisable in order to enable the patient to digest sufficient food. Such cases not uncommonly occur in the course of a progressing pulmonary tuberculosis or of chronic renal disease (chiefly the granular contracted kidney).

Again, when there is permanent damage to the mucous membrane of the stomach, as in the atrophic changes subsequent to catarrh or in the changes that occur in carcinoma ventriculi, the administration of pepsin is advisable, in conjunction with hydrochloric acid. This may also be done in severe cases of dilatation of the stomach without bacterial fermentation; but it is as a rule not the best treatment, pre-digested food being preferable. The administration of pepsin is indeed best confined to those cases, as has been already said, in which there is no recovery from the permanent damage to the secretory mucous membrane. Although in acute and subacute catarrh, the secretion of the gastric juice, both as regards pepsin and hydrochloric acid, may cease, yet it is not good treatment to give pepsin and hydrochloric acid in these cases. By other means, complete recovery of the function is possible, and, indeed, in such cases the object of treatment is to permit as little digestion in the stomach as is compatible with a resting condition, or to permit none at all in the severer cases (see Diet, Chapter XI.). The mucus which is in the stomach is not digested by pepsin under any condition.

It is thus seen that the administration of pepsin in gastric disorder is very limited in its application.

This is much more the case with regard to pancreatin. The indiscriminate manner in which pancreatin has been given in stomach affections, and the extravagant statements regarding its beneficial effects show a total disregard of the properties of the tryptic ferment. " Pancreatin," which as sold commercially is a mixture, and is not given for its action on starch (amylolytic effect), but for its proteid-digesting powers (proteolytic effect). It cannot digest proteids in an acid medium, only slowly does it act on them in a neutral one, but it acts best in an alkaline medium. If pancreatin is given for its proteolytic effect in the stomach (and it can be given for no other action) it is useless to give it if the food during digestion becomes acid by the secretion of hydrochloric acid ; and, moreover, it does not pass into the duodenum, and become active there in the presence of the alkaline bile and pancreatic juice, because it is destroyed in the stomach by the action of the gastric juice, and by the pepsin as much as by the acid. It is, therefore, of no use whatever to administer pancreatin if there is any, even a moderate, secretion of gastric juice during digestion. It may be of value, however, in those cases, already described, of permanent deficiency in secretion of the gastric juice; and it may be administered with alkaline carbonates, and may either aid digestion in the stomach or, passing through the pylorus, aid digestion in the duodenum. It is thus given in the same class of cases as pepsin, but its value in promoting digestion in the stomach can only be a matter of accident, *i.e.* can only occur if there is no hydrochloric acid present; and in those severe and chronic cases in which so little gastric juice is secreted that pancreatic digestion might be supposed to occur in the stomach, a better treatment is to give the patient peptonised food. The secretion of the gastric juice is the great bar to the action of pancreatin in indigestion of food, and it has been suggested that it should be swallowed in capsules of keratin, a substance which is undissolved by the gastric juice but is capable of digestion by the pancreatic juice. It has been found, however, in practice that the keratin capsules, more frequently than not, are passed per rectum unchanged.

The use of pancreatin is best reserved for the preparation of peptonised food.

Papain has been said to have proved of value in the treatment of disease of the stomach, both in children and adults. Its use may be said to depend on the same circumstances which guide the use of pancreatin, with the exception that it is not so sensitive as trypsin to the reaction of the medium in which it acts.

The administration of ferments in gastric disorders has a very limited scope. In ordinary cases they are unnecessary, although there is no doubt that the use of pepsin after meals, even in simple cases of gastric disorder, tides a patient over a period of gastric disturbance. This is, however, a habit which is to be discountenanced, since the cause of the disorder can in most cases be discovered in irregularities of diet or of the mode of life, and can be remedied by rational treatment.

5. *Remedies acting as Sedatives to the Stomach.*—To give rest to the stomach is one of the first axioms in the treatment of acute and even chronic affections of the organ. This is chiefly accomplished by determining what amount of work the organ can do without discomfort, and limiting the diet accordingly. But there are conditions in which irritability

of the stomach, either nervous or motor, has to be treated specially.

Motor irritability is shown partly by spasm of the stomach, and although this may in some cases be treated by antispasmodic remedies (p. 294), yet drugs with a direct sedative action may have to be given. Motor irritability is also shown in acid eructations during digestion, and in vomiting. On the part of the nervous system more particularly, irritability is shown by the presence of epigastric pain, and by the numerous reflex effects of gastric disorder (see Chapter. VI.). If these conditions of irritability are produced by hyperacidity of the gastric contents, the administration of alkalies is the proper treatment, and it is effectual in the early stages. But if the condition be severe, alkalies alone will not relieve, whereas alkalies in combination with sedatives will act beneficially. Irritability, both nervous and motor, is a feature of gastric irritation, gastric catarrh, and chronic ulcer. It is also a feature of "nervous" dyspepsia, and of some cases of gastric insufficiency, but in these cases the stomach condition is secondary to a general condition of the nervous system. In gastric irritation, the condition of irritability is induced directly by food, aided in some cases by a general condition of the body; in gastric catarrh by a continued inflammation or chronic congestion of the mucous membrane; and in chronic ulcer, by the presence of an open sore which is irritated by the food and the acid contents of the stomach, the irritability being also often aided by a general condition, viz. anæmia.

Some of the sedative remedies which are used act on the central nervous system, others act locally; and as regards others an explanation of their sedative action on the stomach is not very clear, although their physiological action on the central nervous system is known.

Of general sedatives, morphine is the chief; it may be used hypodermically in a dose of 2 or 3 minims of the injectio morphinæ hypodermica (B.P.), the dose to be repeated in an hour and a half to two hours, if necessary. Its use in stomach affections is limited to cases where there is great epigastric pain, such as sometimes occurs as an emergency and is not relieved by removing the stomach contents, or to cases where there is repeated vomiting and retching, persistent efforts being made after the stomach is emptied; chiefly, however, in those cases where the vomiting is due to severe irritation of the stomach with or without congestion. In cases of purely nervous vomiting, morphine hypodermically is, as a rule, to be withheld; and if given, a repetition of the dose has to be carefully considered. When morphine is given in nervous vomiting, it is usually in those prolonged cases which are unrelieved by other remedies.

Codeine may also be used in cases similar to those in which morphine may be given; it possesses no advantage over morphine, and is weaker in its physiological action. It is given in doses of $\frac{1}{4}$ grain gradually increased. Except in the treatment of diabetes, it is rarely necessary to exceed a dose of one grain, three times daily.

Of local sedatives to the stomach, ice is of great value. In cases of persistent vomiting due to irritability of the stomach in gastritis, in chronic ulcer, and in other conditions, the sucking of ice gives great relief to the stomach symptoms. Its use is limited to this condition of vomiting, as in gastric uneasiness and pain it gives practically no relief. Its effect is only temporary.

Another means of relieving vomiting in irritation of the stomach, and of the retching which sometimes follows it, is in the form of local applications to the epigastric region. In epigastric pain this also gives relief. A mustard leaf may be placed over the epigastric region, or better still, hot dry fomentations (*i.e.* a four-folded piece of flannel about 18 inches square). The fomentation is to be covered with cotton wool, and the whole kept in place by a firm flannel bandage. In this case not only does the heat give relief, but the bandage gives rest and support to the abdominal muscles and the lower part of the thorax, and aids in relieving the symptoms of retching and pain.

Of local sedatives in the form of drugs, there are three which stand out pre-eminently for their useful action, viz. morphine, hydrocyanic acid, and cocaine. Morphine and hydrocyanic acid, as well as cocaine, are local sedatives; they relieve pain when applied to the part affected. Cannabis indica is inferior to these as a local sedative, but it is useful in many cases. Morphine is best given in 7 to 10 minim doses of the liquor morphinæ hydrochloratis (B.P., containing 1 per cent); hydrocyanic acid is given in 2 to 4 minim doses of the acidum hydrocyanicum dilutum (B.P.); cocaine in doses of $\frac{1}{15}$ to $\frac{1}{10}$ grain and cannabis indica in the form of tincture in 7 to 10 minim doses. Morphine, as a sedative to the stomach, is best given in combination with either alkalies or acids as the case may indicate; hydrocyanic acid is best given with alkalies which hold it in combination in the mixture, and cannabis indica also with alkalies, although it is not incompatible with acids. Cocaine is best given with acid mixtures, or it may be given in pill form. Extractum cocæ liquidum in doses of $\frac{1}{2}$ to 2 drachms may also be used as a gastric sedative; but although it appears to be useful in some cases, its effect in the majority of cases is very disappointing and far behind that of cocaine itself, or morphine, hydrocyanic acid, and cannabis indica.

These sedatives are used to relieve epigastric pain when occurring in the course of the digestion of a meal, to relieve the reflex pain in the chest, and the other symptoms of gastric irritation and irritability. This they do effectually in many instances, when given in mixture three times daily before or after food; the treatment being continued for from three to six weeks. The administration of hydrocyanic acid in such small doses may be continued with benefit for a much longer time than this; but with morphine, cocaine, and cannabis indica, these drugs must be omitted as soon as the sedative effect is obtained. They do not, as a rule, produce any general effects of the drug, but morphine sometimes causes constipation.

Bromide of potassium in doses of 5 to 10 grains, especially in combination with iodide of potassium (2 to 3 grains), is a remedy which frequently has a beneficial sedative effect on the stomach. It is a local sedative, as its action on the throat shows; and it probably acts on the stomach, partly as a local, and partly as a general sedative of the nervous system. If continued for longer than three or four weeks, it, however, is apt to produce depression, both bodily and mental, and, like the iodide, may produce a rash; its action has therefore to be carefully watched. It is best given in combination with alkaline carbonates. The action of iodide of potassium is difficult to explain, but that it has a beneficial effect on the stomach when a sedative is required is demonstrated in many cases of disordered function. Both the bromide and iodide are most beneficial in cases of gastric irritation and are given before meals. Ammonium or sodium bromide (dose, 5 grains) may be substituted for the potassium salt; they are less depressant in their action.

Bismuth, either in the form of nitrate or of carbonate, is frequently given as a sedative to the stomach, especially in combination with hydrocyanic acid. Its effect, however, is more frequently disappointing than clearly beneficial. Both salts appear to have the characters of inert powders, and may thus be said to have a useful sedative action. Their prolonged administration, however, leads to the odour of garlic in the breath, frequently to nausea, and even in some cases to an aggravation of the symptoms of irritation. The carbonate is the preferable salt to administer, and it acts to some extent as an antacid, but its action in this respect is far inferior to the alkaline carbonates of sodium, potassium, magnesium, and calcium. The nitrate as prescribed is usually acid, and its acidity increases on keeping. It not uncommonly acts as an irritant.

Ether and chloroform may be classed together as mild local sedatives to the stomach in the doses which are permissible. In large doses they may act as irritants. Spiritus ætheris may be given in doses of $\frac{1}{2}$ to 1 drachm; and chloroform may be given either as spiritus chloroformi (dose, 10 to 15 minims), or as aqua chloroformi in ounce or half ounce doses. Chloroform is preferable to ether and is better borne as spiritus than as aqua chloroformi. Both are useful adjuncts and correctives to mixtures prescribed for stomach disorders.

Carbolic acid (dose, 1 grain) and *creasote* (dose, 1 minim) are both local sedatives as well as antiseptics, and are given for both these effects in stomach disorders. Carbolic acid is more useful than creasote and is less apt to irritate; it is most conveniently administered as the glycerinum acidi carbolici (B.P.) in doses of 5 to 10 minims. In this form, prescribed with alkalies, it frequently obviates the administration of the more powerful sedatives, such as morphine and cocaine.

6. *Antifermentative Remedies.*—Antifermentative remedies are administered in stomach disorders with two objects: (1) to prevent bacterial fermentation of the food in those conditions of the organ predisposing to it; and (2) to stop fermentation when it has once begun.

The conditions favourable to bacterial fermentation of food in the stomach are three in number (see p. 70):—

1. An insufficient secretion of hydrochloric acid.
2. The delay of food in the organ.
3. Motor insufficiency, showing itself in its most marked form in dilatation of the organ (see Chapter. XIV.).

When hydrochloric acid is secreted in normal or abnormal quantity, bacterial fermentation does not and cannot occur. It is useless therefore to give antifermentative remedies in these cases, as is not infrequently done. The conditions in which hydrochloric acid is deficient and bacterial fermentation likely to occur are:—

1. Long-standing gastric insufficiency.
2. Subacute catarrh and long-standing chronic catarrh.
3. Prolonged dilatation of the stomach from whatever cause.

Part of the treatment of fermentation of food is considered under the heading of diet (Chapter XI.), of washing out the stomach (Chapter XII.), and of dilatation of the organ (Chapter XIV.). Antifermentative remedies are, however, frequently of the greatest service in combination with other modes of treatment.

The following remedies are used as antifermentatives:—

Acidum hydrochloricum dilutum in doses of 10–15 minims.
Acidum nitro-hydrochloricum dilutum in doses of 10–15 minims.
Sodii hyposulphis in doses of 10–15 grains in solution.
Sodii sulphocarbolas in doses of 10–20 grains in solution.
Acidum carbolicum, most conveniently administered in the form of glycerinum acidi carbolici, 5–15 minims in solution.
Creasotum in minim doses in the form of pill.
Acidum salicylicum in doses of $\frac{1}{2}$–2 grains, given in capsule or in solution, much diluted.
Resorcin in 5 grain doses in solution.
Beta-naphthol, an insoluble powder, given in 2–5 grain doses in capsule.

Of these remedies the three most effective are the mineral acids, hyposulphite of sodium, carbolic acid, and creasote. Creasote is apt after a time to irritate, and great care must be taken not to continue its administration for too long a period. Carbolic acid may be given for a long period to prevent bacterial fermentation in the stomach, and hyposulphite of sodium is chiefly used to stop fermentation which has already commenced. Its action is dependent on its decomposition in the presence of acids, by which the antiseptic sulphurous acid is set free. This decomposition is produced not only by mineral acids, but by organic acids, when present in large quantity. Both carbolic acid and the hyposulphite are best given before meals, creasote soon after. Salicylic acid is apt to irritate, and has a very limited application as an antifermentative to the stomach. It is best given towards the end of a meal.

Resorcin is non-irritating and may be continued for a long time; but its action as an antiseptic is feeble.

Beta-naphthol is best given in capsule soon after meals, in order to avoid the nauseous bitter taste which may produce nausea or even vomiting. It is of great value in counteracting bacterial fermentation in the intestines, both small and large, as a large part of the quantity taken passes down the intestinal tract undissolved. Salol is an excellent antifermentative for bacterial fermentation in the small intestine; it is decomposed in the duodenum and upper part of the small intestine into salicylic acid and carbolic acid, which act as antiseptics. It is thus not an antifermentative for the stomach, as the drug itself is practically inert as a bactericide.

The administration of remedies can only in comparatively few cases take the place of washing out the stomach with dilute antiseptic solutions (Chapter XII.), and although in dilatation of the organ remedies are of value, they are subsidiary to washing out the organ. Where, however, it is desired to prevent fermentation, the remedies mentioned are of great service, and even in this case an occasional cleansing of the stomach is of great value. The administration of antifermentatives is the means of treating one form of flatulence, that due to bacterial fermentation in the stomach and intestines, by which large quantities of gas are generated.

Antifermentatives relieve this form of flatulence by stopping the bacterial fermentation. For enabling the patient to bring up or to pass the gas, which frequently accumulates in the stomach and intestines, antispasmodic remedies are to be used.

7. *Emetics.*—Emetics are only in rare instances to be used in gastric affections. Owing to the prostration following their use, they tend to aggravate the stomach symptoms. When it is desirable to empty the stomach in cases of gastric irritation, gastric catarrh, or dilatation of the organ from whatever cause, it is best to wash out the organ, using a soft tube. This produces less general disturbance than the administration of emetics. In some alcoholic cases, on the other hand, an emetic may be the best remedy to use, and the most serviceable is apomorphine. In doses of $\frac{1}{10}$ to $\frac{1}{6}$ grain hypodermically, apomorphine acts as a prompt emetic, and in the majority of instances does no harm. It may, however, produce incoordination of gait and a general depression of the body. It must not be given to old people, to those debilitated by long illness, or those with serious organic disease (cardiac and renal), or to those with a weak circulation.

Mustard and water, one teaspoonful of mustard in a tumblerful of warm water, is a safe emetic. And salt and water, two teaspoonfuls to a tumblerful of warm water, may be used in some cases. But copper sulphate, alum, and ipecacuanha are to be avoided on account of their irritant effects.

8. *Remedies for Constipation and Diarrhœa.*

Diarrhœa.—The four forms of diarrhœa which occur in connection with gastric disorders are:—

1. Diarrhœa directly due to irritant food, and coming on shortly after the food has been taken. The treatment of this is an avoidance of the recurrence of the indiscretion, but if the symptoms are severe (and not due to mineral poisoning) a purgative soon after the commencement of the symptoms is the proper treatment. This treatment must not be delayed longer than about twenty-four hours after the onset of the symptoms; its object is to expel the irritant food from the intestinal tract. If the diarrhœa has lasted some time, it is useless giving a purgative, as the symptoms are now due to

the irritant effects on the intestines, and these must be treated by sedatives and intestinal antifermentatives. The best purgative to administer under the circumstances described is castor oil, of which half an ounce is to be given with 3 to 5 minims of tincture of opium. In this combination no harm is done by the castor oil.

2. Diarrhœa in gastric disorder may be due to bacterial fermentation of food both in the small and large intestines; the motions being foul, and accompanied by the passage of much flatus. A castor-oil purge is frequently a preliminary and great aid in the treatment of this form of diarrhœa, but the administration of intestinal antifermentatives is the essential treatment. Salol (5 grains), beta-naphthol or salicylate of bismuth may be given to adults; and to children, in whom the condition is very common, either salicylate of bismuth (dose, 1 to 3 grains), or hydrargyrum cum cretâ (¼ to 1 grain) frequently repeated.

3. The third variety of diarrhœa in gastric disorder is that which is called lienteric. Lienteric diarrhœa has many causes, some dependent on the condition of the central nervous system, others on the condition of the stomach and intestines. The latter variety is very amenable to treatment. Although it may have existed for two or three months before the patient seeks advice, it is frequently cured in less than a week by the administration of small doses of arsenic before meals; 2 or 3 minims of liquor arsenicalis (B.P.), or 3 to 4 minims of liquor sodii arseniatis, with half a drachm of tinctura lavandulæ composita in an ounce of water. In what manner the arsenic acts it is impossible to say. A second mode of treatment is the administration of gastric sedatives in an alkaline bismuth mixture shortly before meals; either 2 to 4 minims of acidum hydrocyanicum dilutum (B.P.) with 10 to 15 grains of bismuth carbonate, or 7 to 10 minims of liquor morphinæ hydrochloratis with bismuthi carbonas and sodii bicarbonas. These mixtures are of use in many cases, but are not so generally useful as arsenic. The treatment must be continued for three or four weeks, as the condition is apt to recur. Sometimes the diarrhœa is succeeded by obstinate constipation, which has to be treated by mild aperient pills and by diet.

4. A fourth variety of diarrhœa is that which alternates with constipation in the course of a chronic gastric disorder (p. 204). The treatment is that of chronic constipation, since the primary condition is constipation, and the secondary, irritation of the walls of the gut by the hardened fæces and bacterial decomposition of the fæcal matter. The treatment, therefore, is to get rid of this fæcal matter by aperients.

Constipation.—Habitual or chronic constipation is frequently one of the most obstinate symptoms to combat in gastric disorders. It may be due to other conditions and diseases than those of the stomach, but as a rule it is a feature of both acute and chronic gastric affections. The treatment is by no means simple, and is dependent on the regulation of the diet and of the mode of life, on the medicinal and other treatment of the gastric affection, as well as on the administration of aperients. It is a symptom which is one of the troubles of advancing age.

Simple measures are in many cases sufficient to overcome habitual constipation. The habit of going to the closet regularly, and preferably after breakfast, is one of great importance to be observed. It is frequently broken, especially by women, and may lead to a chronic constipation. Smoking after breakfast is an aid, but the custom of rushing off after a hasty morning meal is a hindrance to the normal daily evacuation of the bowels.

Fruit such as oranges, apples, figs, and prunes taken before or, better still, after breakfast is often beneficial. Oatmeal at breakfast or brown bread, instead of white, acts similarly. But in chronic gastric affections, fruit, oatmeal, and brown bread are more frequently inadmissible than not; the fruit leading to organic acidity of the gastric contents, the oatmeal and brown bread containing much indigestible cellulose, which not infrequently irritates both the stomach and the intestinal tract.

The remedies which are given for overcoming habitual constipation are either simple purgatives or purgatives combined with tonics. None of the more violent purgatives are necessary, since the effect required to be produced is a gradual one. As an emergency either enemata or strong purgatives may have to be administered. The continuance of enemata or of strong purgatives does more harm than good.

Enemata.—Small enemata are to be used, not exceeding a pint, either of soap and warm water, or of thin oatmeal made with water. If there is much flatulence, one teaspoonful of oil of turpentine may be added to the oatmeal enema. Glycerine enemata are sometimes useful, either by means of an injection into the rectum of one drachm of glycerine with a glass or vulcanite syringe, or by means of a glycerine suppository, which, being a solid body and slowly melting, is not so serviceable as the injection of glycerine, as it is sometimes pushed out of the rectum before sufficient glycerine has been dissolved to have its proper effect. Glycerine enemata if long continued are apt to irritate the mucous membrane; and in some cases they cause an evacuation every second time they are used and not every time.

Enemata are to be used only in cases of emergency, as an occasional adjunct to the slow action of the aperient habitually given, and in cases where it is not desirable to give any food or medicine by the mouth. These latter cases are those in which there is violent and continuous vomiting, as in acute gastric catarrh and chronic ulcer; where there is severe catarrh of the stomach without much vomiting (as in the patients who are under treatment); where there has been recent hæmatemesis from whatever cause; and where there is great dilatation of the stomach.

Saline Purgatives.—As a rule saline purgatives are only to be used in gastric disorders as adjuncts in the treatment of chronic constipation. Thus in the continuous treatment of this disorder with mild aperient pills, salines given in the early morning before breakfast are of great use in aiding the action of the mild evening pill. Two teaspoonfuls each of magnesii sulphas and sodii sulphas may be given with five or ten drops of tinctura zingiberis in half a tumblerful of warm water. Some of the aperient saline waters may be used: Hunyadi Janos or Æsculap, half to one wine-glassful in half a tumblerful of warm water, sipped while dressing in the morning. The administration of salines in this way ought only to be occasional in gastric disorders. In some patients, they diminish the appetite for breakfast, in others they cause depression. They are, however, of great use, in men especially, and in those who are obese and suffer from chronic gout. Continuous administration of salines is sometimes adopted, in a mixture containing a drachm each of magnesium sulphate and sodium sulphate, 15 to 20 grains of bicarbonate of sodium, and an ounce of infusion of gentian, with a corrective such as spiritus chloroformi or spiritus ætheris (5 to 10 minims). In not a few cases, in women, this alkaline mixture taken before each principal meal has an excellent effect in regulating the bowels. But it frequently produces disagreeable effects and cannot be administered for any length of time. It causes depression, magnesium sulphate being a very depressant drug; and after a time it produces small watery motions, passed three or four times daily. This is not a desirable effect, and soon counteracts any benefit the patient has previously received from treatment; so that if tried, the patient must be directed to stop taking the medicine as soon as the stools become watery and more frequent, and a simple alkaline mixture substituted in order that the medicinal treatment of the case may not be intermitted. Given in smaller doses (10 grains three times daily) sodium sulphate is of service in regulating the bowels, especially in combination with tincture of belladonna.

Mercurial Purgatives.—For the treatment of habitual constipation by the continuous administration of aperient medicines, mercurial purgatives are to be avoided. If given frequently, they do more harm than good. Yet in certain cases, their action is most beneficial. In the first place, they may be given as an adjunct to a mild aperient pill. When in the early stage of treatment, the daily pill is insufficient to produce a regular motion every day, an occasional mercurial purgative is useful. One of the best remedies for this purpose is a pill containing 2 grains of calomel with 2 grains of pilula colocynthidis composita (B.P.), or one containing the same amount of calomel with 2 grains of pilula rhei composita. The patient is to be directed to take this pill occasionally at night, omitting the daily mild pill. In the second place, in certain cases of gastric disorder associated with pains in different parts of the body, such as a soreness of the muscles, a pain in the chest (especially the left side), and more particularly in cases of pain in the neck passing over the head, a blue pill (pilula hydrargyri, 5 grains) at night, followed by a black draught (mistura sennæ composita, 1 ounce) in the morning before breakfast, frequently gives great relief. These are not to be frequently repeated, as by repetition they lose their effect. The administration of small doses of mercurial salts for the relief of constipation is to be avoided in gastric disorders in adults.

Remedies for Continuous Administration in Chronic Constipation.—The remedies previously discussed are those which are to be used mainly in emergencies in chronic constipation. Effectual relief in chronic constipation, however, depends on two chief conditions :—

1. The treatment of the gastric disorder by remedies, hygienic regulations, and by diet.
2. The continuous administration of mild and tonic aperients.

In mild cases, the administration of an alkaline bitter mixture before meals, and a slight alteration in the diet is sufficient to regulate the bowels, which from being opened once in two to four days are opened every day. In other cases, malt preparations or cod-liver oil also aid; and in many cases where these remedies are indicated, an aperient medicine is found unnecessary. But in the majority of cases of chronic constipation in gastric disorder, there is great difficulty in treatment, and an aperient medicine becomes a necessity. It must be one which can be administered every day without bad effects, the object being to produce a normal daily motion; solid, and without any succeeding watery evacuations.

Senna, in any simple mixture, is not suitable for continuous administration; but mixed with a large amount of correctives, and with a small quantity of sulphur as in pulvis glycyrrhizae compositus, it is a very valuable remedy for constipation both in children and adults. It must be given at night, and the dose to be administered depends on the effect. In children of seven to fourteen years, it is best to begin with a daily dose of half a drachm; in adults, of a drachm. If this amount is insufficient, it may be increased to 2 drachms; if too large, *i.e.* producing watery evacuations, it must be diminished. Beyond 2 drachms as a daily dose it is unnecessary to go, and so large a dose cannot be administered for any length of time. In such cases, treatment with liquorice powder is far behind in efficiency the method of a daily mild pill with an occasional stronger one. In some cases liquorice powder cannot be borne, partly from its nauseous taste, which may be overcome by the addition of half a teaspoonful of sal volatile, and partly from the griping produced.

Senna in the form of confectio sennæ is to be given in these cases where there are hæmorrhoids; it must be taken in teaspoonful doses before breakfast.

Rhubarb, Cascara, Aloes.—The three purgative remedies which are par excellence of benefit in chronic constipation are rhubarb, cascara sagrada, and aloes. Of these rhubarb and aloes are the most efficient in the majority of cases, although in some cascara agrees well.

Rhubarb.—For adults the best form of administration is the pilula rhei composita (B.P.) in doses of 5 grains every night before going to bed. If the dose (5 grains) is too large, *i.e.* tends to produce small evacuations after the first solid one, the patient may be directed to shave off a small piece of the pill; if the dose is too small, 10 grains may be given, to be reduced to 5 when necessary. Rhubarb pills are best taken uncoated, and they must be freshly made. Old coated pills are very slow in dissolving or may be passed unchanged.

For children rhubarb is best administered either with an alkaline carbonate (sodium bicarbonate) or with a saline purgative. In the former case for a child of three to seven years of age, 6 grains of pulvis rhei may be combined with 3 grains of sodium bicarbonate in powder, a whole or half a powder being given every night. In other cases it is advisable to administer the rhubarb in the form of a mixture taken three times daily before food, viz. one containing tincture of rhubarb, 5 minims; magnesium carbonate, 3 to 5 grains; glycerine, 10 minims, and water to 2 drachms. To this may with benefit be added belladonna, in the form of tincture, in 7 to 10 minim doses. Sodium sulphate (10 grains) may be substituted for the carbonate of magnesium, but as a rule the carbonate agrees better. The rhubarb and soda powders agree well with children, but if the constipation is very obstinate and continues so in spite of alterations in the diet and the daily administration of the rhubarb powder, small doses of liquorice powder are effectual. The rhubarb and belladonna mixture is very suitable for continuous administration in obstinate constipation due to digestive disorders. The dose of rhubarb and magnesium carbonate must be graduated according to the effects of the medicine.

Cascara sagrada may be administered in two forms, the solid extract and the fluid extract. The fluid extract is the more reliable preparation, and as it is disagreeable to take in solution, may be conveniently given in the form of elixir. The solid extract in doses of 2 to 5 grains is given in the pill form, the smaller dose being for continuous daily administration. The fluid extract is, however, the preparation to be employed, and it may be given in two ways. A daily dose of 15 to 20 minims may be given every night to ensure a morning evacuation, the dose being increased to 30 minims if necessary. It is, however, in many cases better to give it three times daily in an alkaline mixture with belladonna and glycerine, as in this case both the gastric disorder and the constipation are treated. Ten minims of fluid extract of cascara sagrada, with 20 grains of bicarbonate of sodium, 7 to 10 minims of tincture of belladonna, $\frac{1}{2}$ drachm of glycerine and 1 ounce of water are to be given three times daily before meals. A mixture such as this, in conjunction with dieting, is frequently all that is necessary to give complete relief in cases of chronic gastric disorder with constipation.

Aloes and Aloin.—Aloes is one of the remedies most serviceable in the treatment of chronic constipation in digestive disorders. It may be given either in the form of extract (B.P.) or of aloin, which is a crystalline substance obtained from aloes. The dose of the extract is 2 to 3 grains, and of aloin $\frac{1}{2}$ to 2 grains. Aloes is contained in pilula rhei composita, and is one of its active ingredients. For the treatment of chronic constipation aloes in the form of extract or of aloin is usually given in the form of pill combined with strychnine and belladonna, or sulphate of iron, as in the so-called "dinner pills." In the construction of a dinner pill the dose of the purgative must be small, inasmuch as anything like purging is not desirable in the treatment of chronic constipation. The best form of dinner pill is as follows:—

℞.

Extracti belladonæ	gr. ss.
Extracti nucis vomicæ . . .	gr. ss.
Ext. aloes socotrinæ	gr. ii.

To be made into a 5-grain pill with soap or with powdered liquorice root and treacle.

This pill may be modified in several ways. Extract of hyoscyamus, 1 grain, may be substituted for extract of belladonna, in cases where patients are sensitive to the action of belladonna. Aloin, $\frac{1}{2}$ grain, may be substituted for the extract of aloes, and $\frac{1}{2}$ or 1 grain of sulphate of iron added to each pill. Thus a useful pill is composed of $\frac{1}{2}$ grain each of aloin, extract of nux vomica, extract of belladonna, sulphate of iron, powder of myrrh, and soap.[1] Powder of ipecacuanha, $\frac{1}{2}$ grain, is in some cases a useful substitute for the extract of belladonna. Podophyllin resin in $\frac{1}{4}$ grain doses, and euonymin in 1 grain doses, are constituents in some dinner pills, but these are not so generally useful as the pills just described.

Dinner pills are to be taken daily with the last meal, viz. between 7 to 9 P.M. Their action is occasionally aided by an occasional saline draught (p. 310) before breakfast, or by substituting occasionally a purgative evening pill for the dinner pill, either a mercurial and colocynth pill (p. 311) or a colocynth and hyoscyamus pill (B.P.).

On the Use of Alcohol in Affections of the Stomach.—In a pure form diluted alcohol is a stimulant to secretion; when undiluted or in the form of beer or wine it is an irritant to the stomach, causing hypersecretion after a time, but at first greatly delaying the chemical processes of digestion.

In the treatment of stomach disorder, two points must always be borne in mind; (1) as to whether alcohol in any form is to be allowed as an article of diet, or (2) whether it is to be given medicinally. These two conditions must be weighed distinct from each other. In allowing alcohol as an article of diet, the state of irritation and irritability of the stomach itself must be considered, as well as the kind of alcoholic drink to be allowed. Thus in all acute states of the stomach, alcohol in any form given by the mouth is harmful; in acute and chronic gastric irritation, in acute catarrh, and in the subacute exacerbations of chronic catarrh, in irritable conditions of the stomach purely nervous, and in chronic ulcer in young adults, alcoholic drinks do harm and must be withheld. When, however, the acute or subacute condition is past, the blandest forms of alcoholic drinks as an article of diet alone are to be allowed, viz. the ardent spirits much diluted, and taken in small quantities with meals. Afterwards, well-matured claret and champagne are admissible, but not the heavy or acid wines—Burgundy, port, or claret. Allowing alcoholic drinks in these cases is a concession to the patient and not a necessity in the treatment. On the other hand, in not a few cases of gastric disorder, alcohol has to be prescribed as a remedy. It is always best given in stomach disorder as matured whisky or brandy, sometimes as Bordeaux. Port from its acidity is always inadmissible. It is always to be given with food and much diluted, and the amount to be given daily is dependent on the general condition of the patient. As a rule three or four tablespoonfuls daily is sufficient in the treatment of gastric disorders. The cases in which it is to be prescribed are those of gastric insufficiency, and especially when this is permanent. It acts here as a stimulant to secretion and to the nervous system. In cases of ulcer in middle or old age, where there has been no recent hæmatemesis, alcohol is frequently of benefit, although care must be exercised in its use. In cancer also, and in all cases of degeneration of the mucous membranes, it may be given with benefit.

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[1] Sir Andrew Clark, *Lancet*, Jan. 1, 1887.

A word may be added regarding other food accessories taken as drinks. In all cases it is best to proscribe tea, except as a flavouring agent for milk; coffee may be given in the same way. Although cocoa is allowable, its continued use is as a rule objectionable to the patient.

CHAPTER XI.

TREATMENT OF ACUTE AND CHRONIC AFFECTIONS OF THE STOMACH—*Continued.*

TREATMENT BY DIET.

THE principles to be observed in the selection of a normal diet have already been fully discussed (Chapter I.), as well as the varying digestibility of articles of diet and the effect of food accessories on normal digestion (Chapter II.). In Chapter III. food was considered as a cause of disordered digestion, and through all the preceding pages, the relation of diet to gastric disorders has been constantly under consideration.

It now remains to discuss to what extent the diet has to be varied from the normal to suit the altered digestive capacity of the stomach and to prevent the food doing harm in functional and organic diseases of the organ. It must, in the first place, be a paramount object to ensure the absorption into the system of a certain amount of nutriment, which may require to be small, as when the patient is continuously at rest in bed; or may have to be nearly the normal amount, as when the patient, although suffering from disordered digestion, is obliged to follow his occupation. How to accomplish this with the least change of the individual habits is the aim of dietetics in gastric disorder. For acute and severe gastric disorder, the dietetic treatment is, as a rule, simple and easily carried out, since the patient is an invalid, incapable of working; but it is in the chronic cases, especially those with acute or subacute exacerbations, that dietetic treatment is difficult of application and often appears to fail, chiefly because, when the patient feels better, he returns to old indiscretions of diet, with a necessary recurrence of symptoms.

In the dietetics of gastric disorders there are several general points which, first of all, deserve attention, all of which are corollaries of the statements regarding diet previously discussed (Chapters I., II., and III.). There is no such thing as a definite diet for gastric disorders. Even in acute conditions no definite prescription of diet can be laid down as suitable for all cases. This is still more so in chronic gastric disorders, in which great idiosyncrasy with regard to food is met with. In chronic cases, therefore, the patient has to be consulted as to what agrees and what does not agree. The statements made may be fanciful; they often are, but, on the other hand, they are not uncommonly correct, as up to a certain point the patient is the best judge of the discomfort produced by a particular article of diet. Failure in the treatment of chronic gastric disorder is not infrequently to be ascribed to insufficient inquiry of the patient as to the effect of particular articles of diet in producing the symptoms from which he suffers.

The three objects to be borne in mind in the dietetics of gastric disorders are:—

1. To give the patient in the day only just as much food as the stomach can digest and manipulate.
2. To remove the irritants from the diet.
3. To remove the substances which, in cases of bacterial fermentation of the food, are decomposed by bacteria.

On the second, which is by far the most important question in the dietetics of gastric disorders, the first statement to a great extent depends.

1. *To give the Patient only just as much Food as the Stomach can Digest and Manipulate.*—Digestion of the food in the stomach is not quite the same as manipulation. In healthy conditions, as has been shown, the food may, from its character and bulk, be with difficulty manipulated by the stomach, *i.e.* the peristalsis may diminish before chemical digestion is complete, and so the last portions of the meal are not expelled into the duodenum. In disorder and disease of the stomach

insufficient digestion and insufficient manipulation of the food is often present, due either to a deficient secretion of the gastric juice, or to deficient motor activity of the stomach, or to both.

Thus in gastric insufficiency, the conditions to deal with are a deficient secretion of gastric juice and a deficient motor activity, the latter being in some cases the more prominent condition.

In gastric catarrh, acute and chronic, both secretion and motor activity are affected, in the more acute cases even to complete abeyance of the functions of the stomach.

In gastric irritation, a deficiency of function is not present in the early stage, although later a deficient motor activity of the organ may be observed.

In chronic ulcer, the digestion may be normal, or there may be gastric insufficiency or hypersecretion. In cancer there may be gastric catarrh as well as gastric insufficiency. In all cases, except those of gastric irritation, the tendency is to dilatation (motor weakness) of the organ, associated with deficient secretion.

Hence in the majority of cases of gastric disorder the food has to be diminished in quantity; in the acuter cases, it may have to be completely withheld by the mouth. It has to be diminished in quantity by the mouth in the following conditions:—

1. In gastric insufficiency, because of the deficiency in function of the organ.

2. In gastric catarrh, because of the deficiency in function and of the irritability of the organ.

3. In gastric irritation, because of the irritability of the organ.

4. In chronic ulcer, as a means of treatment of the ulcer by rest, or because of the presence of gastric insufficiency or catarrh.

5. In many cases of cancer, on account of dilatation of the organ, and on account of the gastric insufficiency or catarrh.

Diminution in the quantity of the food is not the only point to be attended to: the food articles must be digestible and easily manipulated by the stomach. This has already been fully discussed (Chapter II.), and need only be referred to here.

In the second place, feeding by the mouth may have to be completely stopped, and rectal feeding substituted. The conditions of the stomach in which this is indicated are:—

1. Acute gastric irritation, in some cases of which food must be withheld for thirty-six hours or more to allow the organ to recover.

2. Acute gastric catarrh and some cases of subacute catarrh.

3. In cases of great irritability of the stomach, whether due chiefly to nervous irritability, or to catarrh, or ulcer, in which all food is rejected by the organ, after causing great epigastric pain (see p. 341).

2. *To remove Irritants from the Diet.*—This is the second object to be kept in view in the dietetic treatment of all cases of disease of the stomach, whether functional or organic. An ordinary diet which in health produces no symptoms, in gastric disease is the cause of various symptoms, some of which are directly related to the stomach, others are referred. The mixed food, therefore, acts as an irritant. It is the object of treatment to remove the most irritant substances from the dietary.

In cases of gastric insufficiency, gastric irritation, and chronic catarrh, this removal of the irritant food articles from a diet is in many cases all that is essential in the treatment of the case, when combined with simple medicinal treatment. In other cases, as has just been stated, all food may disagree owing to the irritability of the organ.

The chief irritant substances in the dietary of gastric disorders may again be mentioned (Chapter III.):—

(1) *As regards the Food itself*:—

Too large a quantity of organic acids or salts (acetic, lactic, tartaric, etc.).

An excessive amount of carbohydrates—starch and sugar—and of fats in the dietary.

A large amount of cellulose.

(2) *As regards the Food Accessories:—*

All food accessories are irritants, or tend to become irritants, in gastric disorder, especially when taken in anything like a concentrated form. The most important are alcohol, and especially the acid wines and beer, tea and coffee, pepper, spices, and hot sauces.

In determining, therefore, a diet for cases of gastric disorder, the main points to be remembered are that the food must be bland and digestible; must not contain too much carbohydrate or fat, or too much cellulose; and that food accessories, while necessary for flavouring must be allowed with great caution, in small quantity and much diluted.

3. *To remove the Substances which, in cases of Bacterial Fermentation of the Food, are decomposed by Bacteria.*—This is the third object of dietetics in gastric disorder. In mild cases of fermentation, such as occur occasionally in the course of gastric insufficiency, the carbohydrates of the diet must be diminished in quantity, and in some cases completely withheld from the diet. When bacterial fermentation is, however, continuous and well marked, as in dilatation of the stomach, complete withdrawal of the carbohydrates from the diet is an essential to treatment. All food articles containing starch, much dextrin and sugar are to be withdrawn, at any rate for a time.

From these general considerations, we may pass on to a more detailed discussion of the alterations of the diet to be adopted in the treatment of gastric disorder. From what has been said, it is evident that the diet to be adopted in each particular case of disease or disorder cannot be rigidly defined. Even in organic disease of the stomach, no particular diet can be universally recommended. The diet has to be regulated according to the degree of alteration in the functions of the organ, according to the degree of irritation and irritability of the organ, and according to the nature of the organic disease present—inflammation, degeneration, ulcer, cancer. A practical account, therefore, of the dietetics of gastric diseases is more or less of an empirical nature, a particular change of the diet being indicated by symptoms or groups of symptoms.

Dietetic treatment will, therefore, be considered under the following headings:—

1. Changes of diet in chronic gastric irritation and in chronic gastric catarrh.
2. Changes of diet in gastric insufficiency.
3. Changes of diet in great irritability of the organ: as shown by pain and vomiting.
4. Changes of diet in great flatulence and in acidity.
5. Changes of diet in dilatation of the organ: (1) with or without bacterial fermentation, (2) with or without pyloric obstruction.
6. Changes of diet in organic disease of the organ: ulcer and carcinoma.

1. *Changes of Diet in Chronic Gastric Irritation and Chronic Gastric Catarrh.*—The dietetic treatment of these two conditions may be considered under one heading, since not only does chronic gastric irritation pass into chronic catarrh in many cases, but in both there are symptoms of irritation of the organ. Pathologically, however, there is a difference, inasmuch as in chronic gastric catarrh there is a diminished secretion of gastric juice, especially of the hydrochloric acid, and an increased secretion of mucus; whereas in the majority of cases of gastric irritation, the secretion of gastric juice is not diminished, and there may be an excessive secretion of hydrochloric acid. In these cases dietetic treatment goes hand in hand with the medicinal, which is essential for the relief of the patient. The medicinal treatment may in individual cases be quite different in the two cases, but practically the dietetic treatment is the same.

Patients do not seek advice at the first onset of the symptoms in these conditions, but after a period of one, two, or three months, during which the distress of digestion is gradually increasing, and they come with a history of previous attacks similar to the present. The features of the attack are important from the point of view of dietetics. At first the distress is only after the largest meal in the day, then after each meal, then early morning symptoms come on; tea during the day being found to disagree is given up; alcoholic drinks, although they may disagree, are rarely given up; meat is then eliminated from the diet, sometimes potatoes, until at the time of the first visit patients say that everything disagrees. There may have been occasional vomiting: in the case of gastric irritation the liquid being acid and irritating to the mouth; in that of gastric catarrh, containing mucus but not irritating, unless there is an excess of organic acids in the diet or fermentation is present. The patient, indeed, comes in what may be called a subacute exacerbation of the condition. The dietetic treatment depends on the intensity of the symptoms, and the main question to decide is whether the patient is to be placed on a strictly liquid diet or on a modified ordinary diet. Both these measures are temporary, and with medicinal treatment may give instant relief to the more urgent symptoms. The prescription of a temporary liquid diet depends on the severity of the symptoms of irritation of the organ. It is rarely necessary in cases of gastric irritation, but in cases of chronic gastric catarrh, where the symptoms gradually increase till at the first visit the patient complains that all food disagrees and that vomiting occurs every day or every two days, a liquid diet is an essential to treatment. This may be prescribed in the following forms:—

Strict Liquid Diet.—Cow's milk alone is to be taken; boiled, and cooled to the temperature that is most palatable. Hot milk in such cases not infrequently causes pain, as well as cold milk. A tumblerful of milk is to be taken every two hours regularly day or night, so that three or four pints of milk are taken in the twenty-four hours. The patient need not be in bed, but must rest most of the day, although a short walk of half an hour twice daily is advisable. Some patients can pursue their ordinary avocation, if not laborious, on this diet. Constipation, the only serious trouble, is to be treated as previously directed (p. 309). Beef-tea, not soup, may occasionally be allowed as a change to the milk, and a little coffee in the milk, just to flavour it, is agreeable to the patient.

The duration of the treatment by liquid diet of these chronic cases (which have been aggravated by lack of attention) depends on the results obtained. In all cases of gastric irritation where it is necessary, relief is obtained in three or four days, and the diet may then be modified; in cases of chronic catarrh, the same result may follow, but not infrequently the liquid diet has to be continued for a much longer time—from two to four weeks.

This initial and temporary treatment with a liquid diet gives relief to the distress of digestion and to the vomiting in the majority of cases.

A *modified liquid diet* may now be adopted, and this may be prescribed from the first if the symptoms are not very severe. In the modified milk diet, carbohydrate food is added, and the patient is allowed to divide his food into three meals, to be taken at 8 A.M., 12 to 1 midday, and 5 to 6 P.M. Carbohydrate food is not to be given if there are signs of bacterial fermentation of the food in the stomach, unless this is readily prevented by means of medicinal treatment.

Meat juice is a convenient adjunct to this diet. It is prepared by finely mincing good beef, covering with cold water, and adding a little salt. After standing for one to two hours, the juice may be expressed by means of a lemon-squeezer. Two or three ounces of meat may be used, and previous to mincing, it must be lightly broiled, in order to sterilise the surface.

For breakfast at 8 A.M.: bread and milk, made with the crumb of white bread and passed through a sieve; or milk, boiled with two tablespoonfuls of baked flour, which has been baked till it is quite brown but not burnt.

For the midday meal, 12 to 1, milk and Benger's food, made quite thin, with the white of an unboiled egg beaten up in it, a little salt being added. Arrowroot (Bermuda or St. Vincent) may be substituted for the Benger's food, or one or two tablespoonfuls of meat juice added instead of the egg.

For the evening meal, 5 to 6 P.M., a basin of milk as at breakfast. In between meals the patient may be allowed a small quantity of milk, with a flavouring of coffee or in some cases of tea, and at bedtime a glass of milk, which may be drunk then or during the night. Modifications will have to be made in the diet, according to the results obtained: if it causes distress the amount of solid food must be diminished; if it agrees more food must be added. To some patients, milk in any form is unpleasant, and in others it disagrees. In very few instances, however, is it necessary to withdraw milk altogether from the dietary. For the full milk (*i.e.* the milk containing the cream), fresh skimmed milk (centrifugalised milk) may be substituted and made palatable by flavouring with a little coffee or cocoa. The dietetic value of the skimmed milk may be increased by beating up with it the white of an egg, or adding a tablespoonful of meat juice prepared in the manner described above. It is advisable also to give malted foods (p. 339) with such a diet, and occasionally to allow a little freshly made beef-tea.

The length of time during which the patient takes this modified milk diet depends on its effects; it may have to be taken with modifications for some weeks. During its continuance its effects must be carefully watched, especially if there is any tendency to bacterial fermentation in the stomach, as the lactic acid or other acid fermentation may occur and undo all the good effects of the diet. In this case the amount of milk must be diminished and its place taken partly by skimmed milk and partly by beef-tea. Moreover, it may be found advisable to give as a midday meal either boiled fish or scraped underdone beef-steak, as in the next diet to be described.

Mixed Milk and Solid Diet.—This diet may be considered as a modification of the last. It is to be prescribed either in the graduated dieting of patients who come with the symptoms described, viz. those associated with the subacute exacerbations of chronic gastric irritation and gastric catarrh, or it may be prescribed from the first, without the stages of the liquid or modified liquid diet.

This diet may be given in two forms. The first consists of boiled milk and carbohydrate food, pounded fish or chicken, scraped steak, or beaten eggs, and bread and butter. The second consists of the same, except that the fish need not be pounded nor the steak scraped, and chicken or pigeon may be allowed, with cocoa.

The meals may be distributed as follows:—

1st Diet.—Breakfast at 8 A.M., consisting of milk boiled with bread, baked flour or German rusks.

Midday meal at 12 to 1, pounded fish or scraped steak and bread and butter. The fish is to be boiled, no sauce added, and before being eaten all bones are to be removed, and the flesh pounded in a mortar or passed through a sieve. It is to be eaten warm and a little milk mixed with it. Plaice without the skin, and haddock and sole are the best kinds to use; mackerel, or the heavier fish as cod, turbot, and halibut being inadmissible. Scraped steak is prepared as follows: 3 or 4 ounces of good rump steak or fillet steak are lightly broiled with a little salt, it is then cut and scraped with a knife, so that the red pulp is separated from the shreds of membrane. The scrapings are spread on thin bread and butter with no crust or mixed with fine bread crumbs, a little salt being added. The scrapings may also be mixed with milk, not too hot, and drunk with it.

Evening meal, 5 to 6 P.M.—Bread and butter with a cup of thin cocoa made with a large quantity of milk, the whole being strained to remove the undissolved particles.

In between these meals and during the night, a glass of milk previously boiled and cooled may be taken.

2nd Diet.—This is the same as the first, except that for the midday meal, boiled fish may be taken, without pounding, or the wing of a boiled chicken, or the breast of a roast pigeon. No flavoured sauces are to be added. In many cases, tender steak, especially fillet, not too much broiled, may be prescribed as a change. Milk puddings (made with an excess of milk) and custards are allowable.

With regard to both these diets, it must again be said that the rational method is to feel your way with the treatment; if either diet disagrees, reduce the amount of solid food; if they agree, an advance may be made in the diet. Both these diets are of great use in commencing the treatment of many cases of chronic gastric irritation and gastric catarrh, where the symptoms, although of two or four months' duration, are not severe and there is but little vomiting. They have as their basis the prescription of milk, fish, and bread as the staple articles of food; all green vegetables, potatoes, prepared (cured) foods, and food accessories (alcoholic drinks, tea, etc.) being removed from the diet. These diets have very beneficial results, combined with medicinal and hygienic treatment; the symptoms abate in severity, the food being digested easily. The duration of the treatment is a matter of judgment; by varying the food articles patients can subsist on such diets and even follow their occupation without discomfort. Gradual additions may be made to the diet till eventually a modified ordinary diet is reached.

Modified Ordinary Diet.—A modified ordinary diet may be described as an ordinary diet, which retains the most digestible food articles and excludes the indigestible and the irritant. No rigid rules can be laid down regarding the digestibility or indigestibility of any particular food article in an individual case; for what may be indigestible in one patient with gastric disorder, is digested without discomfort in another. It is from this point of view indispensable for successful treatment to ascertain from the patient the effects of articles of diet in producing symptoms. Again it is necessary to refer to the fact that in a diet there should not be too great a preponderance of one class of food stuffs, and especially of carbohydrates and fats, which not being digested in the stomach are with difficulty manipulated by the disordered organ. The irritants in the food which have to be removed from the diet for the successful treatment of the cases under consideration are:—

1. Those vegetables and articles of diet containing a large amount of cellulose in their composition: brown bread, oatmeal, cabbage, uncooked green stuff.

2. Vegetables containing an irritant active principle, such as turnips, onions, asparagus.

3. Fruits containing a large amount of organic acids, free and combined.

4. Alcoholic drinks for the most part, not only on account of the alcohol, but of the organic acids present.

5. Other food accessories, especially tea, hot spices, and condiments.

6. Lastly, all cured foods, except bacon, on account of their close texture and the irritant bodies with which they are impregnated during smoking, or which develop from incipient decomposition.

Putting aside the idiosyncrasy of the individual patient, a list may be made of food articles and food accessories to be avoided, and those to be taken.

FOOD ARTICLES TO BE AVOIDED.

1. *Proteid Food Articles.*

Beef in many instances, owing to its close texture and the large amount of sarcolactic acid it contains. In certain forms it may be given (p. 326).

Pork in any form.

Fish: eel, cod, turbot, halibut, and all fried, dried, or cured fish.

Twice cooked food, as this hardens the flesh and makes it indigestible.

Made dishes, with rich sauces.

Game of all kinds, *i.e.* all flesh which has to be hung until incipient decomposition makes it palatable.

Turkey, goose, duck.

Tinned and cured meats, prepared meats, meat pasties, brawn; ham in many instances, smoked fish and meat.

2. *Carbohydrate Food Articles.*

Potatoes in many cases.

Uncooked green vegetables: celery, water-cress, lettuce, endive.

Cooked vegetables and other food articles containing much cellulose: cabbage, brown bread, coarse oatmeal.

Pastry and all close-set puddings and sweet biscuits.

Irritant vegetables: onions, shallots, asparagus.

All fruits, cooked or uncooked, except grapes and pineapple, if the juice only is swallowed.

3. *Fatty Food Articles.*

All fats, except cow's butter and the fat in cow's milk.

4. *Food Accessories.*

(*a*) *Alcoholic drinks.*—Strong and acid wines: port and sherry.

All white wines: Burgundy.

All unmatured wines and spirits.

(β) Tea absolutely, except to flavour milk.

Café noir.

(γ) All hot spices and sauces, except mustard and vinegar in moderation.

FOOD ARTICLES ALLOWED.

1. *Proteid Food Articles.*

Beef in certain forms (p. 326).

Mutton.—The inside of a loin chop, a cut off the thick end of a roast or boiled leg. Cutlets without hot sauce or seasoning.

Chicken, wing or breast; preferably boiled.

Pigeon, the breast.

Boiled fish: plaice, sole.

Eggs, lightly boiled; best eaten *à l'Americaine.*

All these articles must be plainly cooked.

2. *Carbohydrate Food Articles.*

Potatoes in some instances, preferably rubbed through a sieve.

Spinach, fresh green peas, occasionally *cauliflower, sea-kale;* all plainly cooked.

Arrowroot, tapioca, sago, ground rice; made into puddings with a large quantity of milk: *baked flour* with milk.

White bread. In some cases plain, unsweetened biscuits.

3. *Fatty Food Articles.*

Butter. Cream in rare instances.

4. *Food Accessories.*

(*a*) *Alcoholic drinks*:—

Matured whisky and brandy, much diluted and taken with meals; up to one ounce daily, in two portions. Never to be taken between meals.

Bordeaux (claret), only matured wines, to be taken once daily (two glasses) with the principal meal.

Champagne, dry, to be taken once daily with the principal meal; one or two glasses.

The amount of alcoholic drink recommended is to be regulated by the habits of the patient; as a rule in the cases under consideration they are best avoided (Chapter X. p. 315).

(*β*) Flavouring agents allowed are a small quantity of bitter almonds, vanilla, and mustard.

It may be found in determining what diet to prescribe to a patient with gastric irritation or chronic gastric catarrh, that the condition is aggravated and kept up by one or two articles of diet, and in the treatment it may be necessary to remove these only from the dietary. Thus it is by no means common for food accessories to be the chief cause of the mischief; the abuse of tea or of beer, and the practice of taking of alcoholic drinks in between meals. It is best indeed in all these cases to remove tea and alcoholic drinks from the dietary. Otherwise, the most important point is the distribution of the daily food into meals, and the regulation of the habits of life.

The food articles allowed are to be taken in meals as follows:—

Breakfast, 8 *to* 9 A.M.—A small basin of bread and milk, or of German rusks and milk, followed by a little fried bacon, or a lightly boiled egg. A change may be made occasionally by having "scrambled" eggs, without seasoning, and a cupful of milk flavoured with a dash of coffee.

Lunch at 1 P.M.—Consisting of plain boiled fish, with white bread, potatoes if they do not disagree, and spinach or fresh green peas. Instead of fish, the wing or breast of a chicken, or the inside of a loin chop, or a cut off a joint of mutton may be taken. For drink, a tumblerful of claret and water, one sherry wine-glassful of the wine; or whisky (one tablespoonful) in a tumblerful of water. A small milk-pudding, made of rice, arrowroot, tapioca, or sago, with much milk, is allowable.

Dinner, 6 *to* 7 P.M.—Consisting of the same as lunch, but with no vegetables and no pudding. A little whisky and water may be taken with this meal.

No food or drink is to be taken after 7 P.M. If the bed-time is after 11 P.M., a cup of warm milk and water with a plain biscuit may be taken at about 10. This may also be taken in the afternoon at about 4, if the necessity arises; but it is better, provided there is no sense of craving or no sense of fatigue for no food or drink to be taken between lunch and dinner.

No alcoholic drink or tea is to be taken between meals, or late at night. After each meal the patient should keep quiet for half an hour or so, and, if a smoker, one pipe or cigar may be allowed after each principal meal. Exercise is to be taken in the morning and in the evening before dinner.

Other points which are important in the daily regulation of the mode of life of those suffering from disordered digestion may be mentioned. The regulations previously mentioned, viz. the regular partaking of meals, the rest after meals, the taking of exercise, the abstinence from stimulant or irritating drinks at and between meals, have as their object to place the stomach under the most favourable conditions for digesting the food which is taken regularly. The lunch and dinner ought in a dyspeptic not to take more than four hours to digest. One other aid to digestion is the eating of meals in congenial company; an aid which is of the greatest importance in those who habitually lead a sedentary and isolated life, or in those whose work during the day is solitary. A change of life like this to the student and to the literary man is often of greater importance than the prescription of medicines (Chapter XII.).

2. *Changes of Diet in Gastric Insufficiency.*—In gastric insufficiency we have to deal with a deficiency of function of

the stomach, both of secretory and of motor activity. The deficiency may be great, so that permanent dilatation of the organ occurs; the dietetic treatment of this condition is treated under that of dilatation (p. 347). The dietetic treatment of cases of gastric insufficiency, other than those associated with great dilatation of the organ, may be considered under two headings:—

(*a*) Of those cases of *temporary gastric insufficiency*, in which the stomach disorder is secondary to some remediable general condition, such as anæmia and chlorosis, the debility following acute febrile diseases, the debility of prolonged lactation, or the general debility produced by the association of prolonged anxiety and work (see p. 211).

(*β*) Of those cases of more or less *permanent gastric insufficiency*, where the primary cause of the stomach disorder is not remediable or permanently remediable by treatment (see p. 214). These primary causes are of various kinds, and are both general or local. The general causes are such as chronic wasting diseases—chronic Bright's disease (granular contracted kidney), pulmonary tuberculosis, malignant disease, and albuminoid disease (due to tuberculosis or syphilis), and chronic suppuration. The local causes are atrophy of the mucous membrane of the stomach whether primary, or secondary to long continued gastric catarrh or to poisons, and carcinoma of the stomach.

Gastric insufficiency being, therefore, either temporary or permanent, the dietetic as well as the medicinal treatment varies. It may be added that some cases of pulmonary tuberculosis and to a less extent of chronic Bright's disease, what may at one time appear to be a permanent gastric insufficiency, is recovered from; this occurs when the tuberculosis becomes quiescent, and in the intervals of the exacerbations of chronic renal disease.

(*a*) *Dietetic Treatment of Temporary Gastric Insufficiency.*—The dietetic treatment of temporary gastric insufficiency must go hand in hand with the treatment of the general condition producing it, and with the medicinal treatment of the stomach disorder. Thus chlorosis and anæmia must be treated, lactation stopped, and general tonic hygienic regulations enforced. The diet to be prescribed is on the same general lines as previously indicated, viz. to give the organ only the amount and kind of food it can digest and manipulate, to remove the irritants from the diet and to diminish the substances which are decomposed by bacteria in the stomach; for in gastric insufficiency the tendency is for dilatation of the organ to occur, and in many cases it predisposes to bacterial fermentation.

As a preliminary, what has been said (Chapter X.) about the effect of alcohol in gastric insufficiency may be repeated. It is perhaps the only stomach condition in which alcohol may be given with great benefit and in which indeed it is a useful adjunct to medicinal and dietetic treatment, inasmuch as it stimulates the functions of the organ. In prescribing it, the same rules have to be observed as in cases of gastric irritation, viz. the administration of none but non-irritating alcoholic drinks, and only with meals and not between. Of the other food accessories, tea is to be strictly avoided, but coffee and cocoa may be used as flavouring agents for milk. As regards the food to be prescribed in the dietary, there is no necessity to repeat what has been said under the heading of the dietetic treatment of gastric irritation and gastric catarrh, as the dietaries there suggested are available for cases of gastric insufficiency. Thus it may be found in mild cases, that a *modified ordinary diet* (p. 327) is digested easily, in others a *mixed milk and solid diet* (p. 325) has to be prescribed, but rarely has a strict liquid diet to be given, unless irritation is added to the symptoms of insufficiency. Irritant articles of diet have to be removed from the diet, as gastric irritation is never produced if due care be taken. Again, the prescription of food containing too much starch or sugar is to be avoided in well-marked cases of the disorder, and when starchy food is given it must be well cooked and not concentrated.

As the general condition improves under treatment, so the diet may be increased, and no rules can be laid down, except the general ones already discussed, for the particular food articles to be added to the diet in these cases. One point must always be borne in mind in the graduated dieting of patients, viz. that if any addition to the diet disagrees by being undigested, it must at once be removed from the dietary and another substituted or the diet be allowed to remain as before.

(β) *Dietetic Treatment of Permanent Gastric Insufficiency.*—In cases where gastric insufficiency is permanent, the primary disorder is incurable, but there are, as has been shown, gradation in the degrees of gastric insufficiency. In pulmonary tuberculosis, in chronic Bright's disease, in cardiac disease, the primary disease may be so much ameliorated that the stomach can recover its functional activity, although in advancing pulmonary tuberculosis, not only may the stomach not recover but the mucous membrane may undergo degeneration, as the result of catarrh. The most typical cases of permanent gastric insufficiency are met with in degeneration of the mucous membrane.

Mild cases of the disorder may be treated by the same diets as those recommended for the curable cases of gastric insufficiency. Many cases, however, have to be treated by a purely liquid diet, or by means of predigested food. It is in such cases indeed that peptonised food is of the greatest use, and that they can be resorted to without doing injury to the organ. The administration of peptonised food must, however, be dependent on the fact that the stomach cannot digest and manipulate sufficient food for the needs of the body, and when it is found that the stomach can digest even a little food it is best to give this along with peptonised food. The latter must only take the entire place of digestible food when the stomach is almost completely inefficient.

Preparation of Peptonised Food.—Peptonised food is made by the action of extract of pancreas on milk or meat, or fine gruel made from various carbohydrates. The mode of preparation is very simple and can readily be learnt by the sick-room attendant. The only difficulty consists in the kind of pancreatic extract to be used. Pancreatic extracts vary greatly in strength, like all extracts of ferments, and it is practically impossible to ensure anything like a uniformity of strength. Sir William Roberts recommended extraction of the pancreas by means of weak spirit, and this was utilised in the preparation of Benger's liquor pancreaticus, which is largely used in the manufacture of peptonised food. The tryptic (proteolytic) ferment of the pancreas acts best in a slightly alkaline medium (equal to $\frac{1}{2}$ or 1 per cent of sodium carbonate); while pancreatic diastase acts well in a neutral medium. Added to this that the temperature of the digesting mixture must be about 40° C. (102° to 104° F.) and the conditions of pancreatic digestion are present, viz. an active ferment, an alkaline medium, and a warm temperature. In preparing peptonised food for administration by the mouth, its flavour must be kept palatable. The action of trypsin on proteids (such as the casein in milk) results in the production of a bitter substance, which has not yet been isolated, and the bitter taste of pancreatised milk is frequently an insuperable bar to its being continuously taken by the patient. It is of no moment if the peptonised food is to be administered per rectum. The bitter taste of pancreatised food may be minimised by watching the action of the ferment during the preparation of the food; the longer the ferment acts and the more active the ferment, the more is the bitter taste developed. The best way, therefore, to proceed is to gauge the activity of the sample of ferment used, by determining how soon a distinct bitter taste is developed in the preparation of the first portion of food by the method to be detailed, and immediately stopping its action by boiling the mixture. Having once gauged the activity of the particular sample of pancreatised extract, the amount to be used and the time during which the ferment is to be allowed to act is known for subsequent preparation. These precautions are very necessary, since they greatly aid the administration of pancreatised food, which may be essential for the wellbeing of the patient.

Pancreatised milk, milk-gruel, and beef-tea may be prepared, and the directions which follow are those given by Sir William Roberts.[1]

Peptonised Milk.—To a pint of milk, add a quarter of a pint of water in which 20 grains of bicarbonate of sodium has been dissolved; heat the mixture to a temperature of 140° F. (60° C.) and add two teaspoonfuls of liquor pancreaticus. Put in a jug and cover with a cosy in a warm place. Digestion will have advanced sufficiently in an hour or an hour and a half; it is then stopped by boiling the mixture. It is best to taste the mixture from time to time, *i.e.* after the first $\frac{3}{4}$ hour, and to boil it directly the bitter taste is distinct, but not excessive.

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[1] "Digestion and Diet," p. 200 *et seq.*, 1891.

A simple procedure, in that it does not involve the use of the thermometer, is as follows: mix the water containing the bicarbonate of sodium with the milk as directed, and divide the mixture into two equal parts, boil one portion and add it when boiling to the other. The mixture is now of the desired temperature, the liquor pancreaticus may be added and the digestion allowed to proceed as in the first case.

Skimmed milk may sometimes be used with advantage instead of the full milk.

For convenience in the sick-room, Sir William Roberts recommends the preparation of pancreatised milk in the cold. The temperature of the sick-room is 60° to 65° F., and the procedure is as follows:—

To a pint of milk is added either half a pint of lime-water or half a pint of water containing 20 grains of bicarbonate of sodium in solution; afterwards three teaspoonfuls of liquor pancreaticus are added. The mixture is placed in a jug and allowed to stand in the sick-room for three or four hours. At the end of this time it is ready to be taken, either by itself or with soda-water. If it is to be kept some time it must be boiled, otherwise it would develop too bitter a flavour.

Peptonised Milk-Gruel.—A thick gruel is made by thoroughly boiling in water, either wheat flour, arrowroot, sago, pearl barley or oatmeal. While still hot add to half a pint of the gruel, half a pint of cold milk in which 20 grains of bicarbonate of soda are dissolved. The temperature of the mixture is about 125° F. (52° C.). Two teaspoonfuls of liquor pancreaticus are now added and the mixture well stirred and placed in a warm place for two or three hours. It is then boiled and strained. During the digestion the mixture must be tasted from time to time to see that the bitter flavour does not become too pronounced. As soon as it is evident, the digestion must be stopped by boiling the mixture. The action of the liquor pancreaticus on the milk-gruel is to convert the casein of the milk partly into peptones, some leucine and tyrosine being formed, and to convert the starch of the gruel into dextrin and maltose, which helps to disguise the bitter flavour developed by the action of the pancreatic extract on the milk.

Peptonised milk-gruel may be utilised in the manufacture of soups, jellies, and blanc-mange, which may be made in the ordinary way, except that peptonised milk-gruel (after boiling) is used as the solvent of the gelatine and to mix with the stock for the soup.[1]

Peptonised Beef-tea.—Sir William Roberts gives the following directions for the manufacture of this food:—

"Half a pound of finely minced lean beef is mixed with a pint of water and 20 grains of bicarbonate of soda. This is simmered for an hour. When it has cooled down to a lukewarm temperature a tablespoonful of the liquor pancreaticus is added. The mixture is then set aside for three hours, and occasionally stirred. At the end of this time the liquid portions are decanted and boiled for a few seconds."

The resulting liquid contains 4·5 per cent of organic residue, three-fourths of which are peptones. Flavoured with salt it is said to resemble in flavour ordinary beef-tea.

The nutritive value of peptones has been established by repeated experiments, in which the ordinary proteids of the diet were replaced by peptones, and it was found that the animal gained in weight. From this result the nitrogen of the peptones must be considered as existing in a form available for the nutrition of the tissues. Experiments with feeding kittens with peptonised milk also demonstrate the same fact (Roberts).

The manner in which the peptonised foods are to be administered is that observed in giving a liquid diet, viz. in small quantities. The actual amount given must be judged by the tolerance of the food by the stomach, or by the extent to which the food is palatable to the patient. The great objection to peptonised milk is that it is not pleasant to take, so that in some cases its utility is very circumscribed. As a rule 4 ounces of peptonised milk may be given every two hours, amounting to nearly 2½ pints in the twenty-four hours.

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[1] Roberts, *op. cit.* p. 204.

Half the quantity, 2 ounces, may in some cases be given every hour, so as not to have too much liquid in the stomach at a time. Two or three times daily, 4 ounces of peptonised milk-gruel may be substituted for a similar quantity of the peptonised milk. As a rule, however, if the stomach is capable of digesting and manipulating undigested milk, this ought to be given as well as the peptonised food. It is not judicious too readily to assume that the stomach is incapable of doing any work and prescribing solely a diet of predigested food, for although in peptonised milk all the casein is not transformed into peptones, some being left to be acted upon by the pepsin-hydrochloric acid, yet the action of the gastric juice on the casein in peptonised milk is not the same as that on full milk. Milk, although taken in a liquid form, becomes in the stomach practically a solid food, since the casein is rapidly precipitated in mass by the curdling ferment and by the hydrochloric acid. This is an advantage to the disordered stomach, since it is a greater stimulant to the motor activity of the organ than food which remains liquid in the stomach. It is therefore advisable to give, if possible, undigested milk as well as peptonised food in the cases under consideration. One advantage of peptonised food is, that being taken in an alkaline condition (*i.e.* containing bicarbonate of sodium), it acts as a stimulant to secretion, and this again constitutes another reason for administering digestible food with the peptonised foods.

In the majority of cases the administration of peptonised foods must only be considered as a temporary measure. This is so in gastric catarrh and other acute cases of stomach disease (see p. 341), also in most of the cases of gastric insufficiency. In cases, for example, of severe pulmonary tuberculosis and of chronic Bright's disease, a partial recovery from the stomach disorder is very frequent, and is aided by the administration of peptonised foods. These, however, must be diminished or altogether withdrawn when it is found that the stomach is tolerant of digestible, and not solely of predigested, food. In cases of pernicious anæmia and of atrophy of the mucous membrane of the stomach, the administration of peptonised foods may have to be continuous, and when intolerance to food by the mouth supervenes, they have to be administered per rectum.

Malted Foods.—Malted foods are those in which vegetable diastase has been allowed to act on starch and dextrin, from which maltose is formed. The essential constituent of malted foods is maltose, which is the sugar formed from starch by the salivary and pancreatic amylolytic ferments (diastases). When a predigested starchy food is required to be given, it may be administered in the form of the peptonised milk-gruel described above. Maltose may also be given in a concentrated form, as in the substances sold as "maltine" or "extract of malt." These are said to possess the additional advantage of containing active diastase, which, although it is inactive in the acid stomach contents, yet passes into the duodenum and completes the digestion of starch there, *i.e.* aids the pancreatic diastase in its action. Whether this be so or not, it is certain that in cases of gastric insufficiency these preparations are useful, chiefly because they are maltose preparations, containing indeed a substance which is of great use in the bodily economy and one which is readily absorbed by the mucous membrane. Both preparations are apt, in some individuals, to produce looseness of the bowels.

Prepared Fatty Foods.—One of the great difficulties of dietetics in disease is to administer fatty foods in a form in which they are tolerated by the stomach and readily absorbed in the small intestine. In disease, carbohydrates in the form of dextrin and sugars, especially maltose, are prescribed to avoid this difficulty, since carbohydrates can to some extent usefully replace fats in a dietary.

A digestible fat may be described as one obtained from an animal, not vegetable, source, and one containing only a small proportion of free fatty acids. The best fats are therefore fresh cream, rapidly separated (by the centrifuge) from recently drawn cow's milk; and cod-liver oil, expressed from the liver of the fish soon after death and purified. The fat of the different sorts of meat is indigestible because of the shreds of tissue mixed with it. For administration in gastric disorder, fresh cream and cod-liver oil are the only two fats admissible. Cream more frequently disagrees than not; partly because it is rarely fresh, at any rate in large towns, and partly because it is readily decomposed by bacteria. Cod-liver oil is the better fat to administer in gastric disorders. The reasons that cod-liver oil disagrees are not the same in all cases. In some it has to be withdrawn because it produces nausea, from its smell and taste. This is obviated partly by taking the oil in an aromatic mixture such as with peppermint water, or with bitters such as infusion of quassia; but chiefly by using only the best kinds of oil prepared from the perfectly fresh livers of the cod. In other cases the oil disagrees because it remains in the stomach a long time and becomes decomposed, causing disagreeable eructations tasting of the oil. This may be obviated by prescribing the oil with strychnine and quassia or with ether, whereby the movements of the stomach are stimulated and the oil more rapidly expelled into the duodenum. A mixture containing 1 or 2 drachms of oil, with 5 minims of liquor strychninæ and an ounce of infusion of quassia, may be given twice daily after meals; or 20 minims of ether may be given with 2 drachms of oil. The strychnine mixture is preferable to the one containing ether, and is less apt to upset the patient.

Cod-liver oil is frequently given in the form of emulsion. Bicarbonate of sodium (like other alkalies) forms an emulsion with it, as also does the alkaline pancreatic juice. But such an emulsion does not facilitate to any great extent the absorption of the oil, unless the stomach contents are not acid. If they are acid the alkali is neutralised and the oil is set free from the emulsion. In cases where the stomach contents are not acid, the administration of oil as a rule disagrees, owing to the great diminution in functional activity of the stomach. A nearly neutral emulsion is what has to be aimed at. Given intimately mixed with maltine or extract of malt, cod-liver oil is frequently well borne in cases of gastric insufficiency, a small dose being given at first and a gradual increase being made.

Mode of Administration of Malted and Prepared Fatty Foods.—It is impossible to state dogmatically in what cases it is advisable to administer these foods, and in what cases they will be tolerated by the stomach. In gastric disorders, they are, as a rule, only tolerated in chronic cases and in those not associated with bacterial fermentation. They are given to supplement the deficiency of fat and carbohydrate in the diminished diet prescribed. Thus in many cases where a modified milk diet is prescribed, the addition of extract of malt or of maltine in 2 drachm doses twice daily after meals is beneficial; also when a mixed liquid and solid diet is given, both maltose preparations and cod-liver oil are of benefit. In both these diets there is a deficiency of fat in the dietary, and for the treatment of the general condition, the assimilation of fat is essential to the progress of the patient.

The dose of cod-liver oil to be given is from 1 to 2 drachms, commencing with 1 drachm once daily after the principal meal or at bedtime, and increasing to twice daily if the smaller dose does not disagree. As a rule, it is not advisable to give more than 2 drachms twice daily, and in but few cases of gastric insufficiency can this dose be tolerated by the stomach.

3. *Changes of Diet in Acute Conditions in which from Inflammation, Irritability or other Causes, Digestion does not take place or is greatly Diminished, or there is Persistent Vomiting.* —These conditions may be classified as follows:—

1. Cases of acute inflammation of the stomach:

   (*a*) Acute gastric catarrh.
   (*β*) Gastritis toxica.

2. Cases of excessive irritability of the stomach, in which all or nearly all the food taken into the stomach is persistently vomited, or causes great pain in the stomach region. Such cases occur in acute inflammation of the organ, in chronic ulcer, in neuroses of the stomach, and in hysteria. All these cases may be grouped together, inasmuch as the stomach is in a state of great irritability, as shown either by pain or by persistent vomiting. The class of cases in which this symptom is present in disease elsewhere than in the stomach is not now under consideration, such as cases of acute cerebral affection, of renal disease, of intestinal obstruction, and of peritonitis or abdominal tumour.

Some of these emergencies in gastric disorder may be treated medicinally with great benefit, so that the persistent vomiting is controlled and the pain relieved. But in any case, unless great care be exercised in the diet, the symptoms return, and dietetic treatment must, in all cases, go hand in hand with medicinal.

For the dietetic treatment of such cases there are two alternatives :—

1. Feeding by the mouth with liquids in small quantities; milk, beef-tea, peptonised foods.

2. Feeding by the rectum with the same liquids, or by means of nutrient suppositories.

The adoption of either method depends on the question whether the liquid food can be retained by the stomach without causing disturbance. When it is not so retained, rectal feeding must be resorted to.

1. *Feeding by the mouth* with small quantities of liquid is to be adopted either at once or after a period of treatment with rectal feeding. Many of these cases when first seen are at their acme, having from one cause or another become aggravated. It is best therefore to begin with a period of one or two days' rectal feeding before giving any food by the mouth. This is the case in acute gastric catarrh, in excessive vomiting and pain, in chronic ulcer, and in neuroses of the stomach.

When feeding by mouth is begun, three foods may be used —milk, beef-tea, and peptonised milk.

Milk.—The milk is to be boiled before use. Cow's milk is usually used; but asses' milk may in some cases be tried. From 1 to 2 ounces of milk are to be given every hour regularly. If this disagrees, $\frac{1}{3}$ part of water may be added to the milk and a little salt.

In cases of vomiting the milk is to be given cold, or even iced, and in all cases it should not be given hot. In persistent vomiting, it is sometimes useful to add lime-water to the milk, in the proportion of 1 in 4, *i.e.* one tablespoonful of lime-water to three of milk. Lime-water is to some extent a sedative to the stomach, but its chief action when administered with milk is that it makes the clots of casein which are formed in the stomach much finer than they otherwise would be, and thus more easily digested. London milk is also always slightly acid, and lime-water neutralises this slight acidity.

Feeding by milk in this manner is very useful after the first two or three days in acute catarrh, also in chronic ulcer of the stomach and in persistent vomiting and severe epigastric pain.

The actual amount of milk given hourly must depend on the effect on the patient. If it disagrees, it may either be diluted, as previously stated, or it may be given at two-hour intervals. In some cases skimmed milk will be found to agree better than the full milk.

An increase in the amount given must be made very gradually. When the two-hourly intervals of feeding are reached, two tablespoonfuls of milk may be added to each alternate feeding. The next step is to add two tablespoonfuls of milk to each feeding, making the total amount of milk taken in the twenty-four hours to about 36 ounces.

As a rule, the patient may now be fed in the manner previously described with a liquid diet (p. 323), and the food gradually increased according to its results on the patient to a modified liquid diet (p. 324), and then to a mixed milk and solid diet (p. 325), and a modified ordinary diet (p. 327). The increase must, however, be very gradual.

Beef-tea may be used as an adjunct to the milk diet, when the patient can take 4 ounces of milk every two or three hours. Not more than half a pint of ordinary beef-tea is necessary in the twenty-four hours. It acts chiefly as a stimulant and is beneficial from the amount of salts it contains.

Peptonised Milk.—If milk cannot be taken, peptonised milk may be substituted and given in a similar manner. It is sometimes advisable to give milk and peptonised milk alternately, and this sometimes succeeds better than giving the latter as the sole diet. As soon as possible a pure milk diet ought, however, to be resorted to. The administration of peptonised food must be looked upon as only a temporary measure in these cases; its prolonged administration when ordinary milk can be digested prevents the recovery of functional activity of the stomach.

2. *Rectal feeding* may have to be adopted in any of the cases

under consideration; in acute gastric catarrh and in gastritis toxica, because it is important to give the stomach complete rest on account of the acute inflammatory condition and the cessation of digestion. In persistent vomiting, and in great epigastric pain caused by food, because in the one case no food is retained in the stomach, and in the other rest to the organ is necessary for the relief of the pain. In most cases rectal feeding is only to be adopted when no food can be taken by the mouth; but in some cases, when the amount of food taken by the mouth is very small, it may be supplemented by rectal feeding.

In acute gastric catarrh, the treatment is best commenced by the withdrawal of all food by the mouth, only a little iced water being allowed to relieve thirst; and during the few days (2 to 7) of abstinence from food, rectal feeding is necessary to support the strength of the patient.

In gastritis toxica, rectal feeding is imperative, and has to be continued for a long time if the patient survives the irritant effects of the poison. The actual period during which rectal feeding must be continued in these cases is determined by the cessation of retching, and by the effects of small quantities of liquid food (not hot) tentatively administered.

In persistent vomiting, due to gastric disorder and uncontrolled by sedatives or by the first diet recommended (p. 342), rectal feeding must be resorted to, and continued until the symptoms cease. Such cases occur chiefly in subacute gastric catarrh, and in chronic ulcer of the stomach. The cases of epigastric pain, in which rectal feeding is to be recommended, also occur in cases of chronic ulcer; rarely in cases of "neuroses" of the stomach is rectal feeding necessary, but it sometimes has a salutary effect on the condition of the nervous system of such patients.

Mode of Administration.—Before commencing rectal feeding, the bowel must be emptied, if necessary, by a simple warm enema of a pint or a pint and a half. After an hour or two the first nutrient enema may be given. Not more than 4 ounces of injection must be given, and when the rectum is intolerant a smaller quantity must be administered, although in some cases irritability is relieved by adding a few drops of tincture of opium to the injection, or by the occasional use of a belladonna suppository (gr. ¼). The injections are not to be given more frequently than every four hours, night and day, and are to be made with a 4-ounce indiarubber syringe, to the nozzle of which a soft indiarubber tube 4 inches long is attached for insertion into the rectum. The injection must be made with the patient lying on the side, and made very slowly. If there is any tendency for the liquid to return, the injection must be stopped for a time, and then slowly proceeded with. By a slow and gentle injection, irritability of the rectum is not often produced. The rectum readily absorbs peptones, and even undigested liquid foods such as milk, beef-tea, and eggs beaten up in the liquid.

Food used in Rectal Feeding.—These are of two kinds, undigested and predigested.

Undigested Foods.—Milk, beef-tea, and defibrinated blood are given. As a rule in the conditions under which rectal injections are given, sufficient milk cannot be given in the enema to sustain life. It is, no doubt, only slowly absorbed. A useful injection to commence with consists of milk, 3 ounces, and beef-tea, 1 ounce. Every second injection the yolk of an egg is to be added to the mixture and well beaten up, a pinch of salt being also added. As a more nutritious material than beef-tea, defibrinated blood may be given, 1 ounce being added to the 3 ounces of milk in each injection, or it may be given by itself instead of the milk. It is more easily absorbed than milk or egg, and contains a larger proportion of proteid food than milk (from 4 to 6 grammes per cent).

Extract of malt may be dissolved in the injection, one teaspoonful in each. It is easily absorbed, and is an advantageous means of increasing the amount of carbohydrate food given.

Predigested Food.—There can be no question that predigested food is more readily absorbed from the rectum than undigested. Peptones are readily absorbed by the rectum, and the conditions for absorption in the thin liquid of peptonised milk and milk-gruel are very favourable.

Peptonised milk (p. 335) or peptonised milk-gruel (p. 336) may be administered in amounts of 2 to 4 ounces every four hours. In the preparation of the peptonised food, the action of the ferment may be allowed to proceed for a slightly longer period than previously recommended, since the bitter taste, which is the disagreeable feature of taking the food by the mouth, is no longer of any consequence. The nutritive value of the enema may be greatly increased by beating up an egg in the milk before the liquor pancreaticus is added. The portion of milk containing the egg must not be boiled before the ferment is added.

The preparation of the peptonised food may be obviated by simply adding two teaspoonfuls of liquor pancreaticus to 3 ounces of milk-gruel (p. 336), and 1 ounce of beef-tea before the injection is made. Bicarbonate of sodium (20 grains) may also be added. The digestion goes on in the intestine and the digestive products are absorbed when formed.[1] Yolk of egg may also be added to this enema, and defibrinated blood or peptonised beef-tea (p. 345) may be substituted for beef-tea.

Peptone suppositories are useful adjuncts to the administration of nutrient enemata, especially in cases where the rectum becomes intolerant of the continued injection of liquid. Given in between the injections, they are also of use, as the proteid food they contain is in a form which is readily absorbed. They are best made with peptonised beef, which is obtainable as a dark-brownish paste with a very bitter taste. Each suppository weighing about 60 grains, contains 50 per cent of the beef peptone.

4. *Changes of Diet in Flatulence and in "Acidity."*—But little need be said under this heading, as the dietetic treatment of these conditions is included for the most part in the directions previously given. When flatulence and acidity are due to bacterial fermentation in a dilated stomach, the carbohydrate food must be removed from the diet (see par. 5, below). But in the other cases we have to deal either with gastric irritation or with gastric insufficiency or catarrh; hyperacidity being a common symptom in the first condition with or without flatulence, but one which is not present in gastric insufficiency and gastric catarrh, in which flatulence may be excessive.

The dietetic treatment of gastric irritation associated with hyperacidity and with flatulence is the same as that previously discussed (p. 322), the acidity being treated medicinally by antacids, and the flatulence by antispasmodics. Certain articles of diet, however, lead more particularly to these symptoms, or aggravate them when present; these are foods containing an excess of organic salts or acids, such as green vegetables and fruits of all kinds, all indigestible foods (such as cold meats, cured meats, and fish), which stay a long time in the stomach; and lastly, all alcoholic drinks, even whisky and brandy, and tea. So that in acidity and flatulence, associated with gastric irritation, these articles of diet must be rigidly excluded.

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[1] Roberts, *op. cit.*, p. 128.

In cases of gastric insufficiency and gastric catarrh where flatulence is not infrequently a prominent symptom, it may be produced by the articles of diet just mentioned, but inasmuch as in both these conditions the tendency is to dilatation of the organ, and to the development of bacterial fermentation of food owing to the diminished secretion of hydrochloric acid, the treatment of flatulence is more medicinal than dietetic, and calls for the administration of antifermentative remedies. Carbohydrates must in great part be withheld.

5. *Changes of Diet in Dilatation of the Stomach.*—In dilatation of the stomach, of whatever origin, there is great weakness of the motor power of the organ, and a diminution of the secretory activity; therefore the food has to be greatly diminished, and the diet must consist of only the most digestible articles. Indeed, in the majority of instances, resort must be had to the simplest of diets. In addition, however, to the diminution of function, there may be bacterial fermentation of the food, and obstruction of the pylorus, which increases the difficulty with which the food leaves the organ. Diet in these cases has to be regulated (1) according to the degree of diminution of function, (2) according to the presence or absence of bacterial fermentation, and (3) according to the presence or absence of pyloric obstruction.

Moderate Cases of Dilatation.—These occur in cases of gastric insufficiency and of chronic gastric catarrh, and the diet suitable in their treatment has already been fully discussed (pp. 332 and 322). It must be remembered, however, that inasmuch as bacterial fermentation is always liable to occur when the stomach is dilated, carbohydrates must be given sparingly, and in some cases withheld.

Great or Permanent Dilatation of the Stomach (see Chapter XIV.).—In these cases, when treatment is commenced, the change in diet must be regulated by the degree of disease, or by the effects of medicinal (antifermentative) treatment, and of washing out the stomach with antiseptics (Chapter XII). There is a choice of three modes of feeding :—

1. By the mouth, in cases where the bacterial fermentation is controlled by remedies.
2. By the stomach tube, after the organ has been washed out.
3. By the rectum with nutrient enemata.

In the majority of cases when at the first visit of the patient there is great bacterial fermentation and great dilatation of the organ, or great dilatation with inflammation (as in subacute catarrh), it is best to begin the treatment by rectal feeding with the nutrient enemata previously recommended (p. 345). The result of the rectal feeding in these cases is very beneficial, the signs of irritation and irritability of the stomach subside, the dilatation becomes less marked, and the patient improves in his general condition. The duration of the rectal feeding cannot be specified ; it may be only for a few days or it may be for a month. In any case, when the stomach is prepared for receiving food, either by the results of the treatment or by remedies applied to cure the bacterial fermentation, feeding by the mouth must be begun very gradually and in the manner described under the dietetic treatment of gastric insufficiency (p. 332). The food must be increased according to the increase in the digestive powers of the patient.

In all cases when food is given by the mouth in dilatation of the stomach, the danger of bacterial fermentation must be borne in mind : therefore the feeding must be supplemented by antifermentative remedies, or by washing out the stomach. In many cases of non-obstructive dilatation a useful method of treatment is to wash out the stomach at the end of the day, so as to remove the unabsorbed remains of the food : during the day the patient is fed on liquid diet. Another method is to wash out the stomach in the morning, and to give food by the tube before it is removed, the patient being fed in the ordinary way by the mouth during the rest of the day, not, however, receiving much food after 8 P.M. In pyloric obstruction, and in some obstinate cases of non-obstructive dilatation rectal feeding is the chief method for giving the patient food, and if it is well borne it is of great benefit to the patient. This has frequently to be resorted to in cases of cancer of the pylorus and in cases of advanced bacterial fermentation, in which the introduction of the stomach tube causes great distress.

Dieting in chronic dilatation of the stomach may be summed up by saying that rectal feeding is the chief support on which to rely for giving the patient a sufficiency of food; and that feeding by mouth must be done with great caution, and always in combination with measures for the counteraction and cure of bacterial fermentation of the food. After urgent symptoms have disappeared, feeding by the mouth must be begun with care, small quantities of liquid food, or of solid food in a digestible form, such as pounded fish, scraped steak, and minced meat balls being given.

6. *Changes of Diet in Ulcer and Carcinoma Ventriculi* (See also Chapters XV. and XVI.).

(*a*) In *chronic ulcer of the stomach* the particular diet ordered is determined with the object of giving rest to the organ, so that the ulcer may heal; either a complete rest or a partial rest. The diet in ulcer may also have to be directed to the relief of the functional disturbances of the stomach associated with it; in the early and active cases there is hyperacidity and gastric irritation, with perhaps great irritability of the organ and severe pain; in the later stages there is gastric insufficiency with sometimes great dilatation of the organ, or there may be chronic or subacute catarrh.

Complete rest by diet is given by rectal feeding, and it is to be prescribed in four chief conditions:—

1. Where there has been a recent hæmatemesis.
2. Where there is great epigastric pain following the ingestion of food.

3. Where there is excessive irritability of the organ, as shown by persistent vomiting and retching.

4. Where the amount of food given by the mouth and capable of being digested by the stomach without pain is insufficient, and so retards the recovery of the patient.

In this last case mouth feeding is very efficiently aided by means of peptone suppositories. As soon as possible feeding by mouth is to be commenced, and in a manner similar to that described previously (p. 342). The food given by the mouth must be either milk, peptonised milk, or milk-gruel, with occasionally beef-tea. When there is dilatation of the stomach in ulcers of long-standing, the same dietetic rules as in other cases of dilatation must be observed (par. 5, p. 347).

(β) In *carcinoma of the stomach* there is again a choice between rectal feeding and feeding by the mouth with a liquid or solid diet. The prescription of rectal feeding is to be guided by the presence of subacute catarrh (p. 341), of great dilatation of the organ (p. 347), or bacterial fermentation. In all cases of carcinoma ventriculi as they progress there is, as a rule, an increasing deficiency in the function of the organ, so that the dieting is essentially that of permanent gastric insufficiency (p. 334), and towards the end rectal feeding is the only means of prolonging the life of the patient.

CHAPTER XII.

TREATMENT OF ACUTE AND CHRONIC AFFECTIONS OF THE STOMACH—*Continued.*

1. *General Hygienic Treatment.*—Throughout the foregoing pages the general hygienic treatment which has to be adopted is indicated ; it may here be summed up. General hygienic treatment has to do with the regulation of the mode of life of the individuals who, although otherwise healthy, are apt to suffer from attacks of indigestion of food (gastric irritation), usually ascribable to some definite indiscretion of diet, or of individuals who suffer from chronic indigestion of food (gastric irritation, gastric insufficiency, and chronic catarrh), *i.e.* of those who are usually termed chronic dyspeptics. In all the conditions mentioned the stomach disorder may or may not be associated with disease, functional or organic, elsewhere than in the stomach, and the mode of life has to be adapted to the condition of body produced by such disease, and according to whether this disease is affected or not by treatment. Thus the hygienic regulations in cases of indigestion of food in pulmonary tuberculosis have to be modified according to the degree and progessiveness of the lung disease, as regards exercise, the amount of food to be taken, and its influence on cough, on diarrhœa, etc. In cases where attacks of indigestion of food are liable to supervene, or where there is no complicating general organic disease, the patient must observe certain rules of life in order that his existence may not be a burden to him ; and, indeed, the adoption and strict observance of such rules for a period of months or years lead ultimately to a cure of the tendency to indigestion of food.

Regular and digestible meals, regular hours, regular and pleasurable exercise, are the three cardinal factors in the warding off of attacks of indigestion of food. The first has already been fully discussed in all its bearings, but it may be well again to emphasise the fact that a large proportion of attacks of indigestion of food are due directly to the food accessories, and especially to alcoholic drinks, to tea, and to hot condiments. In not a few cases abstention from these adds greatly to the pleasures of existence of dyspeptics, since it keeps off attacks of indigestion of food.

Regular hours are as essential as regular and digestible meals. No definite rule can be laid down as to what regular hours are, but such patients as are now under consideration ought not to retire late to bed, and ought to have at least seven or eight hours' sleep. Occupation is an important factor in warding off attacks of indigestion of food. The morning is the time for the hardest portion of the day's work; the midday meal is to be followed by a period of rest, and the afternoon's work should be of a lighter character than the morning's. As regards the evening, a variation has to be made according to the time of life; for as age advances and middle age and old age is reached, the capacity for evening work diminishes, especially in those whose work is chiefly intellectual; but in youth and manhood after the evening meal is frequently the best period of the day for intellectual work. Recreation is essential in the distribution of the day, both simple amusement and intellectual recreation, *i.e.* a change of work from the heavier to the lighter. In all cases, society during meals is of a great advantage; food is not only eaten more slowly and thus is better digested, but conversation also aids digestion often by relieving the mental tension produced by the day's work.

Regular Exercise.—Exercise to do much good to the sufferer from chronic indigestion must be regular and must be pleasurable. Forms of apparatus have been invented by which patients can obtain exercise for the various muscles in their own house. These apparatuses are, however, of no great value: they are all very dull, and patients soon tire of using them In women getting on for middle age, who suffer from indigestion of food and pains in various parts of the body, mechanical exercise as afforded by these instruments is beneficial, but if other exercise can be taken, no mechanical apparatus ought to be used.

As regards the kind of exercise to be taken, this must depend on the inclination and means of the patient. Any form of exercise which is not too prolonged so as to lead to great fatigue and thus loss of appetite may be allowed, and prolonged exercise which interrupts the ordinary meals is harmful. The morning after breakfast, and the evening before dinner, are the best times in the day for taking exercise for those who are closely applied to work all day. As regards the evening exercise, which is usually a walk, the patient not uncommonly says that he is too tired to take exercise. If not yet arrived at middle age, this sense of fatigue which comes from the daily occupation is actually relieved instead of aggravated by the exercise before dinner, so that while beginning sometimes with a sense of being unable to walk, the period of exercise ends with a sense of relieved fatigue and with an appetite. This is much better than taking alcohol and bitters before meals, a custom which is in all cases prejudicial to the patient. In other cases, a short period of exercise, followed by a period of rest, is a good preparation for the evening meal, especially for those of middle age or past it.

A complete change in the mode of life is sometimes of great benefit to patients who, although benefited by treatment, do not recover rapidly. This is not uncommonly seen with those who lead too regular a life, often associated with a monotonous diet. A change to a greater variety of food or to more pleasurable surroundings, is of great service in the treatment. A sea-voyage and a temporary residence in a mountainous district may be of great advantage to such individuals, and no doubt the greater part of the good that sometimes follows a stay at one of the health-resorts at home or abroad is due to the change of scene associated with the idea of going to a place with a definite object, viz. to recover the health, so that the prescribed regimen is carried out by the patient. The recommendation of a voyage or of a health-resort must, however, be carefully considered, special regard being paid to the temperament of the patient; for to many, especially those advancing in years and those who have led a secluded life, a residence abroad is repugnant and may do great harm. The majority of such patients can be treated as well in their own homes as out of them. In deciding upon a locality to which to send a patient, one situated some distance above the sea-level must be chosen, and one in which there is suitable accommodation in the way of lodging and of food.

The last point to be mentioned is one of the most important, viz. attention to the teeth (see Chapter III.). In so many individuals does the condition of the teeth both lead to and aggravate indigestion of food, that an examination of them ought always to be made. The fault may be either that chewing is too painful owing to dental caries and swelling of the gums, or that the teeth are absent to a greater or less extent. In both cases, the condition is easily remedied by the dentist. The neuralgic pains, too, of dental caries are aggravated by disordered digestion, so that both conditions may be cured by attention to the teeth.

2. *Treatment by Massage and by Baths.*—*Massage* is of value in some cases of gastric disorder, and it may be applied in two ways, either as local kneading of the stomach or as general rubbing.

In *local kneading* of the stomach, the object is to increase the motor power of the organ by kneading it in the direction of left to right, *i.e.* in the direction in which the food naturally passes out of the stomach. It is performed by the nurse or other attendant standing on the left side of the recumbent patient and placing the palm of the right hand flat on the abdomen near the left hypochondriac region. The hand is then pressed firmly and deeply into the abdomen and the fundus of the organ, so to speak, gripped in the palm of the hand; this at any rate is the object. Without removing the hand from the abdomen, kneading movements are then made slowly towards the right hypochondriac region, passing over the lower epigastric and the upper umbilical regions. When the right hypochondrium is reached the process is repeated, always passing from left to right. It must be continued for five minutes each time, and afterwards, if necessary, prolonged to ten minutes, and it may be repeated two or three times daily according to results. Such a simple mechanical process is readily learnt by an untrained assistant, and is of great value in many cases of chronic indigestion of food. It is of value in cases where there is delay of food in the organ, and in cases of weakness of the muscular coat (atony) and simple dilatation. It is best applied 1½ to 2½ hours after a meal, and in this case it not only increases the power of the organ to manipulate the contained meal, but it tends in time to restore the motor activity of the stomach.

It is a useful adjunct to medicinal and dietetic treatment in the following cases:—

1. In gastric insufficiency of moderate degree, unassociated with any organic disease elsewhere.
2. In cases of great flatulence in women and in middle age where there is retention of gas in the stomach causing great distress. In these cases, it is usefully combined with kneading of the whole abdomen.
3. In cases of gastric insufficiency, in the later stages of gastric irritation where there is moderate dilatation of the organ, and especially in cases where gastric insufficiency is prolonged in the convalescent stage of acute febrile diseases.
4. In the convalescent stage of acute or subacute gastric catarrh when the stomach is recovering its functions. It is not to be applied if there are symptoms of irritation of the organ.
5. In cases of great dilatation of the stomach it may be tried, provided there is no great pyloric obstruction. It is not, however, as a rule, of much value in these cases.

It is contraindicated by the presence of severe symptoms of irritation of the organ, and must not be employed in cases of ulcer of the stomach, of very painful carcinoma, of recent hæmatemesis, and only with great care when there is cirrhosis of the liver or cardiac disease.

General kneading of the abdomen is sometimes of use in chronic constipation.

General massage, or a systematic rubbing of the limbs and trunk with passive movements of the joints is of value in certain cases. Its object is to increase the power of the skeletal muscles, which it appears to do, partly by influencing the local lymphatic and venous circulations. It is a useful adjunct in the treatment of stomach affections in some cases in which anæmia, but mainly bodily weakness and wasting are present. Thus it may be used in cases of gastric insufficiency which are not associated with serious organic disease; in cases of chronic indigestion of food (not due to organic disease), in which the patient is flabby and somewhat wasted; in cases of slow recovery from chronic gastric catarrh. It must be carefully applied, for it is at first very fatiguing to the patient. It is well to begin with five or ten minutes rubbing once daily; to increase this slowly, until the patient can bear it for ten or fifteen minutes without fatigue; and there must be a period of rest after each rubbing. The results are sometimes good, sometimes disappointing, and are most obvious when muscular wasting and flabbiness require to be treated, and in cases of neuroses of the organ. Massage is of no use as a treatment by itself, it must be combined with medicinal and dietetic treatment directed to the cure of the stomach condition.

Weir-Mitchell Treatment.—Only a few words need be said regarding this mode of treatment, since it is of use not so much in diseases of the stomach, as in disorders of the nervous system which affect the general nutrition. The three essential elements of the treatment are :—

1. *Isolation* of the patient from friends and away from home.
2. *General massage*, daily performed.
3. *Overfeeding*, commencing with a milk diet which is greatly, but gradually, increased; solid food up to three large meals daily being gradually added to the milk diet.

The treatment is applicable to cases of so-called "neurasthenia" in women; such cases also occur in men. The "dyspeptic" symptoms from which such patients suffer are practically limited to flatulence and constipation. The patients are usually greatly emaciated, but they may be stout and flabby.

Baths are to be used as an adjunct to other measures in the treatment of stomach affections, and are of service chiefly for their general tonic effect. Cold baths in the morning are beneficial, but if the patient is unable to stand the cold bath, remaining cold afterwards and having no appetite for breakfast, or if the patient is a child, the bath is best administered in the following way:—A lukewarm bath (at a temperature of about 80° F.) is first taken for about two minutes, and while in the bath cold water (at a temperature of 50° to 60° F.) is poured over the body, or only down the back. The body is then rapidly dried and well rubbed with a rough towel until the skin glows. Young children or weak persons may be rubbed in front of a fire. Breakfast ought to be taken soon after the bath, and it is best to give to children directly after the bath a small drink of warm milk. Adults and children can from this modified cold bath be gradually educated up to taking nearly cold baths with great benefit to themselves.

Cold sea-water may be used instead of freshwater, but as a rule bathing in the open sea is not beneficial to patients with chronic stomach affections.

The other kinds of baths are not so serviceable as the cold bath or the modified cold bath; either of these is readily practised at home, and needs no visit to a special establishment.

3. *Treatment by Electricity.*—The application of the electric current to the stomach is another adjunct to the medicinal and dietetic treatment of chronic stomach affections, which is of service in some cases. The direct application of the faradic current to the mucous membrane of the stomach causes a localised circular contraction of the organ and the secretion of a small quantity of gastric juice. In the treatment of stomach affections, both the constant and faradic currents are used, and they are applied in two ways. By one method (internal or intraventricular electrisation), an electrode is introduced into the stomach, and the other either held in the hand or placed over the stomach region. By the other method large flat electrodes are placed, one over the stomach region, and the other in the vicinity (external electrisation of the stomach).

Intraventricular electrisation is best performed by means of Ewald's modification of Einhorn's instrument, viz. by means of a hollow sound, containing a coiled wire ending in a metal electrode. After the patient has swallowed a glass or two of water, the electrode is passed, and connected either with the negative pole of the constant battery or with one pole of the faradic. The other electrode is held in the hand, or placed over the stomach region or in the rectum (Ewald). With *faradisation* of the stomach, the current must be strong enough to cause contractions but not pain; each sitting is to last ten minutes, and the free electrode is to be moved over the surface of the abdomen. With *galvanisation*, the strength of the current used is from 15 to 20 milliampères, and the sitting is to last about eight minutes. The negative pole is to be attached to the stomach electrode, and the positive pole is to be moved over the stomach region (Einhorn).

External electrisation is performed by means of large flat metal electrodes, or by large sponge electrodes; von Ziemsen recommends one electrode to cover 600 square centimetres, and one to cover 500. The larger electrode is to be placed in the direction of the long axis of the stomach, and with it the negative pole is attached; the smaller is to be placed near the first, but on the left side of the abdomen. The sitting is to last ten minutes, and the current must be strong enough to cause powerful contractions of the abdominal muscles without causing pain.

Uses of Electricity.—The application of the electric current, especially the faradic, in the manner described, has been recommended in the treatment of dilatation of the stomach and of "neuroses" of the organ, chiefly severe gastralgia. In dilatation of the organ, by the continued action of electricity it is hoped to restore the motor activity of the organ; and the method of treatment has been found of use in some cases of dilatation, especially when this is moderate in degree, or when it is associated with "nervous dyspepsia." In great dilatation of the organ it does not appear to be of much avail, and, indeed, so conflicting are the accounts of the effects of the application of electricity in stomach diseases, that its real utility is difficult to determine. For the relief of pain in nervous dyspepsia, it is of value, and in these cases the external application of the faradic current to the stomach region causes a comfortable sensation of warmth in the epigastrium, and tends to increase the appetite.

The application of electricity is not to be regarded as in itself a curative agent in diseases of the stomach, but rather as an aid to the medicinal and dietetic treatment adopted. Its advocates are apt to speak too strongly of its utility.

3. *Washing out the Stomach* (*lavage*).—It is in certain cases of great benefit to the patient to wash out the stomach with regularity; but the method of treatment has been much abused, and must be applied with circumspection.

(a) It is of use in cases of dilatation of the stomach, with or without pyloric obstruction, and with or without bacterial fermentation of the food.

In dilatation of the stomach, there is great delay of food in the organ, which collects for a day or two until it is vomited; the stomach is never empty, and the presence of the food therefore causes great distress, which ends in profuse vomiting. This is prevented by a daily washing out of the organ with a large amount of liquid. When to this delay of food bacterial fermentation of the carbohydrates is added, the distress is still greater, and the need for washing out the organ more peremptory.

(β) It is also of use in cases where there is no great dilatation of the organ, but where a meal causes great distress with epigastric burning, acid eructations, and occasional vomiting. Such cases occur, as has been described, in the exacerbations of gastric irritation, and at the commencement of treatment a daily washing out of the organ for a few days is of great benefit. The patient, especially these with "nervous" symptoms, however, must not be taught this as a means of cure, since the continued use of the practice detracts from the efficacy of medicinal and dietetic treatment.

(γ) In some cases of nervous dyspepsia, associated with great pain (gastralgia), washing out the organ is of value.

(δ) In cases of catarrh the question of washing out the stomach frequently arises, especially in the subacute cases. In the initial stage of treatment this may be done once, and the treatment by rest and rectal feeding proceeded with. When food is again given by the mouth, the question of washing out the organ must be reconsidered, and decided according to the conditions discussed under dilatation of the organ (par. *a*).

The patient may be taught to perform the operation for himself, but, as a rule, only where there is dilatation of the organ associated with bacterial fermentation of the food.

The Time and Frequency of the Procedure.—In all cases the operation has only to be done once daily; in some, it may be performed every two days, and as the patient improves at longer intervals. It may be done either in the early morning or in the evening after the day's food. Whether the treatment is begun in the morning or the evening, eventually it does nearly as much good to the patient; for after a time, the morning washing enables the stomach to perform its duties, as far as it can, during the day when food is taken, and the evening washing acts in a similar way. On the whole, however, an evening washing is to be preferred, and for the following reasons:—The remains and accumulations of undigested food are removed from the stomach, and thus there is not the danger of the food fermenting or causing distress during the long period of rest at night. The presence of this food in the stomach all night frequently leads to restlessness and sleeplessness. Its removal, therefore, tends to give the patient a better night, and he wakes up in a better condition to digest his daily food.

Whether performed in the morning or evening, washing out of the stomach must be done at regular intervals, and must each time be followed by a period of complete rest in the recumbent position.

Liquids to be used.—The water used for the washing out of the stomach must have been previously and recently boiled and cooled to the proper temperature, about 90° F., before being used. Six to eight pints are to be used at each washing, and in this may be dissolved the following substances:—

Boric Acid, 3 per cent (4 drachms to the pint).
Permanganate of potash, added till the liquid is a light pink colour.
Bicarbonate of sodium, 2–4 per cent (3–6 drachms to the pint).
Common salt, 1 per cent (about 1·5 drachm to the pint).

These are the most useful and the least harmful solutions to employ. Other substances are also used: Carlsbad salts, thymol, resorcin, creolin, salicylic acid, etc., but they have no advantage over the foregoing. In some cases astringent substances have been found of use, especially in ulcer of the stomach, viz. subnitrate of bismuth (10 to 20 grammes in 200 cc. of water) and silver nitrate solution (one litre containing a gramme). The latter is applied after a previous washing out of the organ,[1] and is to be removed in a short time, dilute salt solution being given to relieve any sense of burning caused by it.

Method of Washing out the Stomach.—The simplest apparatus is the best. A soft stomach tube is to be used (p. 153), and is to be connected by means of a small piece of glass tubing with soft indiarubber tubing about 2 feet in length attached to a glass funnel, 4 to 6 inches in diameter at its widest part. The stomach tube being passed, the organ is filled with liquid through the funnel, which is done by pouring down two to three pints of liquid. Before all the liquid has run from the funnel, pinch the indiarubber tube near the funnel and lower it, on releasing the tube the stomach contents will flow into the funnel and so into a vessel placed below. When it has stopped flowing, raise the funnel again and pour in more liquid, until six or eight pints have been used. If the liquid does not flow from the stomach a particle of undigested food has probably blocked the tube; it may be cleared by detaching the funnel and blowing down the tube with a ball syringe such as is used for the administration of enemata. When all the liquid has been used and as much removed by siphonage as possible, there is still some liquid in the stomach if it is a case of dilatation. This residual liquid must be removed, and this is readily accomplished by detaching the funnel and expressing the stomach contents by pressure on the epigastrium, the patient being in the recumbent position and on the left side.

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[1] Boas, "Diagnostik u. Therapie der Magenkrankheiten," p. 307, 3rd ed., 1894.

Another apparatus which is more useful than the first is nearly as simple. A siphon tube is attached to the tube from the funnel or irrigator by means of a Y-tube. The irrigator or funnel has a stop- or pinch-cock, as well as the siphon tube. The latter is closed and liquid allowed to run into the stomach from the irrigator to two or three pints; the irrigator tube is now closed and the siphon tube opened, the liquid flowing from the stomach. As soon as the flow has ceased, the siphon tube is closed and the irrigation again allowed to proceed, and so on. After the fluid has ceased running from the stomach, the portion which remains must be expressed, as described with the first apparatus. In some cases it is best to use the stomach aspirator with a soft stomach tube (p. 153). When patients are allowed to do the operation themselves, the first method described is the best for them to employ. It is simplest, and no accident is likely to happen if the connection of the glass tube with the stomach tube is firm. (For other precautions, see Chapter. V. p. 154.)

CHAPTER XIII.

BLEEDING FROM THE STOMACH: HÆMATEMESIS—MELÆNA.

Bleeding from the stomach is shown either by hæmatemesis, the vomiting of blood, or by melæna, the passage of blood in the motions. When blood is present in the stomach, from whatever source it has come, some may be vomited and some passed through the pylorus and so with the motions. But although in the majority of cases of profuse hæmatemesis, there is also melæna; this may be present when there has been no hæmatemesis, and may be due to one or other of the causes mentioned below (p. 371).

Hœmatemesis.—Hæmatemesis is a general term applied to the bringing up of blood from the stomach, and is not confined to cases where the source of the bleeding is the stomach itself. Clinically, there are two chief conditions to be considered, viz. (1) whether the blood vomited has been swallowed, as it not infrequently is, or (2) whether the stomach itself is the source of the hæmorrhage. But the question in individual cases is still further obscured in the following manner: a patient brings up a large quantity of blood through the mouth, the practitioner is called to see him, and the question to decide is threefold, whether the blood has been vomited or not, whether if vomited it comes from a lesion of the stomach or from a lesion in the thorax or above, and if not vomited whether it comes from the lungs, or another part of the thorax. The causes of hæmatemesis may be discussed under the two headings given above.

1. *The Blood Vomited may be Swallowed.*—In this case, the blood comes either from the nose, ear or buccal cavity, from the lungs or from the œsophagus.

In *fracture of the base of the skull* and in other injuries of the head, blood may pass into the stomach and be afterwards vomited.

In *epistaxis*, a similar event may occur, especially if the bleeding be profuse and the patient be in the recumbent position, or if the bleeding occurs chiefly from the posterior part of the nasal cavities. In the profuse epistaxis of some cases of chronic Bright's disease this occasionally occurs, as well as in other forms of bleeding from the nasal cavities.

In *bleeding from the lungs*, if profuse and slow, swallowed blood may be vomited. Cases occur, however, which at the first onset are very difficult to recognise, as when the early tubercular patient has a profuse hæmoptysis associated as it not uncommonly is with vomiting. At the first onset, this may appear like hæmatemesis, but is readily recognised (see Diagnosis, p. 372).

In *bleeding from the œsophagus*, blood may enter the stomach. In acute inflammatory conditions of the gullet, due almost solely to swallowed poisons, this may occur, but in such cases a similar condition of the stomach is present (see below). In cancer of the gullet, the bleeding is as a rule insignificant, and only small quantities of blood are brought up, except in those cases where the aorta is opened by the growth, when the bleeding is profuse and fatal. Lastly, profuse and fatal bleeding occurs when an aneurysm of the aorta opens into the gullet.

2. *The Blood Vomited comes from the Stomach itself.*—The many conditions in which the stomach is the seat of hæmorrhage in hæmatemesis may be divided into three groups, viz.—

(*a*) Where the bleeding is due to disease of the stomach itself.

(*β*) Where it is due to a tumour or growth attached to the stomach.

(*γ*) Where it is due to a general disease of the body.

(*a*) *Hæmatemesis due to Disease of the Stomach itself.*—In this case the hæmorrhage may be of two kinds, either capillary or due to the opening of a larger vessel—vein or artery. The former produces slight bleeding; in the latter, which usually occurs where there is an ulcerated surface, the hæmorrhage is profuse.

Venous or mechanical congestion is a not infrequent cause of hæmatemesis. The pyloric area of the stomach is more readily affected by venous congestion than the cardiac for reasons previously given (p. 229), and when bleeding occurs the mucous membrane may to the naked eye be entire, or it may show numerous small pittings (p. 229) even in the cardiac area, or erosions, as when catarrh is also present. The causes of venous congestion of the stomach leading to hæmatemesis are:—

Dilatation of the right side of the heart, such as occurs as the result of morbus cordis, especially of mitral disease. Hæmatemesis is not a frequent symptom in cardiac disease, chiefly because the liver intervenes in the venous circulation between the stomach and the right side of the heart. When hæmatemesis does occur it is usually observed in the later stages of long-standing mitral disease, where the liver has become fibroid änd fatty, and in this case it is not uncommonly associated with anatomical changes in the mucous membrane of the stomach due to catarrh ("Stäuungs-katarrh").

In direct *obstruction to the portal circulation* hæmatemesis is more common and is often profuse. If the obstruction is acute, as in portal thrombosis, the hæmatemesis is an early symptom, and is often very profuse; if the obstruction is chronic, hæmatemesis is a later symptom and may also be profuse. Chronic portal obstruction occurs in cirrhosis of the liver, and in pressure on the inferior vena cava or on the vena porta itself by tumours or enlarged glands. The two last conditions are rarely the cause of hæmatemesis, so that it may be said generally that acute portal obstruction as a cause of hæmatemesis is due to portal thrombosis; that chronic portal obstruction as a cause of the bleeding is due to cirrhosis (atrophic) of the liver.

Active congestion or inflammation of the mucous membrane of the stomach is a cause of slight hæmatemesis. In cases of gastritis toxica the initial hæmorrhage is due to the erosion and destruction of the mucous membrane by the poison; if recovery takes place, the later hæmatemesis is due to the presence of an ulcer, caused by the effects of the poison. In acute non-toxic catarrh, hæmatemesis is usually slight, and is apparently due to two conditions. The commonest cause is the presence of an erosion of the mucous membrane (p. 257), which leads to slight bleeding; a less common cause is a slight lesion of the softened and congested mucous membrane caused by the presence of indigestible food or by the passage of the stomach sound. There are some cases of catarrh which readily bleed, and although the hæmatemesis is slight, yet its occurrence is important as of diagnostic significance.

Ulceration is the commonest cause of hæmatemesis. In chronic ulcer it is a common symptom (p. 429), and is due either to the opening of a large vessel, such as a branch of the splenic, pyloric, or coronary arteries, or to the rupture of capillaries.

In cancer hæmatemesis occurs when the growth becomes ulcerated; it may be profuse, as when a large vessel is opened, but the bleeding is usually slow (see p. 480).

Excessive vomiting and retching without the presence of arterial disease of the stomach causes slight hæmatemesis, such as occurs in sea-sickness and in some cases of pregnancy. The blood is very slight in amount, only occurring in streaks in the vomit. It is capillary in origin.

(β) *Hæmatemesis due to a Lesion of the Stomach Wall.*—This may occur by the rupture of a neighbouring aneurysm, usually of the cœliac axis or of one of its branches. The aneurysm becomes adherent to the stomach wall, and frequently causes an ulceration of the mucous membrane of the stomach (pressure necrosis) through which it ruptures. The hæmatemesis is sudden, profuse, and fatal.

In other cases a neighbouring malignant growth, usually retro-peritoneal, sometimes omental, invades the stomach, protrudes internally, and ulcerates, capillary hæmorrhage occurring.

(γ) *Hæmatemesis due to a General Disease of the Body, i.e.*

to Diseases of parts other than the Stomach.—Hæmatemesis may occur in—

Acute Febrile Diseases.—In the acute stage of tropical malarial diseases it is a frequent symptom. In typhus fever and in the hæmorrhagic forms of variola and of scarlet fever, hæmatemesis may occur, as well as in the various forms of "septic" fevers and in pyæmia.

In *anæmic diseases*, such as pernicious anæmia and leucocythæmia, and in scurvy, profuse menorrhagia and hæmophilia.

In *renal disease* hæmatemesis sometimes occurs; only, however, in the granular contracted kidney and usually in the stage of high arterial tension.

Vicarious Menstruation. — The occurrence of vicarious menstruation is much disputed; it is strongly upheld by some, altogether repudiated by others. The difficulty in deciding the question appears to rest on the fact that accurate observation of the patient for long periods, during which the character of the menstruation and of the bleeding elsewhere is observed, is wanting. But the fact is indisputable that these patients say that when the menstrual flow of blood is deficient, they have epistaxis, hæmoptysis, hæmatemesis, to mention only three of the kinds of bleeding; and at the same time direct observation has convinced the medical attendant that there is no organic disease present to account for the symptom which ceases with the period.

Of the causes of hæmatemesis considered as due to a general condition of the body, the only one which is of importance in relation to diseases of the stomach is renal hæmatemesis. It may therefore be of value to record a case illustrative of this condition.

A man, aged thirty-six years, came to the hospital with a history of twelve months' illness, characterised by epigastric weight after food, relieved by vomiting. Morning vomiting had existed for over eighteen months, and was not associated with any more definite symptoms than weight after food. One week before his visit to the hospital he had vomited blood three times, altogether about a pint. He suffered from dyspnœa and had been wasting twelve months. He was very pale, with slightly baggy eyelids, but had no other signs of œdema. The appetite was good, the tongue was pale but clean, and the bowels constipated. The pulse showed a slight increase of tension, and the left ventricle was slightly enlarged. On examination of the abdomen, the stomach was found moderately distended, and there was a large area of slight tenderness over the epigastrium which disappeared after rest in bed. The symptoms which suggested that this was not a case of gastric ulcer simply, was the long history of morning vomiting and of dyspnœa without well-marked symptoms referable to the stomach, as well as the increased arterial tension; and the case was definitely recognised as one of renal hæmatemesis by the examination of the urine, which was acid, of low specific gravity (1014), and contained from one-fourth to two-fifths albumin; and by the examination of the eyes, in both of which there were signs of albuminuric retinitis and hæmorrhage, as well as of slight optic neuritis.

Characters of Hæmatemesis.—The chemical and microscopical characteristics of the vomited matters in hæmatemesis have already been discussed (Chapter V., p. 123). The bleeding into the stomach may be slow or rapid; but the vomiting of the blood is sudden, and in the majority of cases the occurrence of bleeding into the organ is unsuspected until the blood is seen in the vomit.

The cases which more usually concern us are those occurring in the course of stomach affections; *i.e.* in mechanical congestion, in acute or subacute catarrh, in ulcer and in carcinoma of the organ. The two commonest causes of profuse hæmatemesis are portal obstruction (chiefly cirrhosis of the liver), and ulcer of the stomach. In these cases, although only small quantities of blood may be vomited, from simply streaks in the vomit to 2 or 3 ounces of clots, yet the blood is frequently as much as one or two pints or more, mixed with the stomach contents. The blood may be a brightish red or dark red and in clots, or it may be "coffee-grounds" in character if it has remained long in the organ. Recurrent hæmatemesis in these diseases is not an uncommon symptom, recurrence after years it may be. The whole of the blood is not vomited at one time, but at intervals of some hours, or, it may be, a day or two; and in these later attacks the blood is frequently darker in colour and may be like coffee-grounds, owing to its long sojourn in the stomach. In cancer profuse hæmatemesis may occur, but as the bleeding is usually slow, the blood is greatly altered and is of the colour of coffee-grounds.

In acute and subacute catarrh slight hæmatemesis is not an uncommon symptom; it is rarely above 2 ounces, and usually consists in a small quantity of blood diffused through the vomit. In chronic cases of catarrh with dilatation this may also be observed; and in most cases, whether acute or chronic, the bleeding is due to an erosion of the mucous membrane. The blood is not much altered in character, and "coffee-grounds" vomiting does not occur.

Symptoms.—The symptoms produced by bleeding from the stomach depend on the amount of blood lost, and on the nature of the disease producing the bleeding. Thus the symptoms of the primary disease may be so serious as quite to overshadow those produced by the hæmatemesis; such, for example, as when this occurs in the course of the acute febrile diseases mentioned, and of profound anæmias, or in the later stages of cancer of the stomach. In these cases hæmatemesis is only a symptom added to a profound change in the body generally; one indicating a loss of blood which perhaps the patient cannot withstand, but which does not add very materially to the already grave signs of illness shown by the patient.

On the other hand, profuse hæmatemesis occurring in the course of cirrhosis of the liver, of chronic ulcer of the stomach, and of rupture of an aneurysm into the stomach produces definite symptoms ascribable to the bleeding, and is not infrequently preceded by a drink debauch, by the partaking of an indigestible meal, or by some sudden exertion.

The patient feels suddenly faint, or gradually becomes faint and pale, with a sense of warmth, sinking, or of actual pain in the epigastrium. This is succeeded by a general sense of coldness of the skin, which may be moist with a cold sweat. The pulse is increased in frequency, it is small and compressible; afterwards it becomes more full and bounding, but is still compressible. Vomiting may occur at any period of the symptoms, the vomit containing blood and food. The patient may suddenly feel faint and vomit the blood immediately, or the faintness may continue without vomiting, but with an increase of the general symptoms, and it is only after a time that the vomited blood shows the cause of the symptoms. After the vomiting the patient usually experiences some relief to the symptoms, although in other cases the general condition is serious, the patient lying partially collapsed in bed with a very pale drawn face, and a very frequent, small, and compressible pulse. These severe symptoms indicate either a continuance of the bleeding, or they are the effect of a profuse loss of blood in a patient previously worn out with chronic illness.

Blood, as previously stated, may be vomited once, twice, or even three times in the same attack, and there may be melæna as well.

In cirrhosis of the liver as well as in chronic ulcer the above initial symptoms of hæmatemesis may be noted. In chronic ulcer, which more particularly concerns us here, after the initial symptoms have passed, there is a stage in the majority of cases of reaction. While the bleeding has left the patient blanched and with bloodless lips and mucous membranes, and has somewhat depressed the temperature of the skin and of the internal parts, the signs of reaction are shown in the recovery of the pulse. During the hæmorrhage and just afterwards the pulse is very frequent, small and compressible; reaction is shown in the pulse becoming more full and bounding, and eventually slowing. The full, bounding pulse is more frequent than usual, and it may be some days before it becomes of normal frequency. During the stage of reaction the patient not infrequently shows a slight pink flush on the cheeks, especially if there has been fair health previously, and the temperature of the skin and internal parts rises slightly, although as a rule not to a febrile extent. The reaction stage after hæmorrhage is more marked in those patients who are least run down by the previous illness. In chronic ulcer, however, after a long period, often years, of illness, there may be no perceptible reaction, and the hæmorrhage may end in death; but as a rule, hæmatemesis in ulcer is not immediately fatal.

The symptoms of slight hæmatemesis are trivial, such as occurs in cases of catarrh and in capillary hæmorrhage in ulcer. A little faintness, soon passing away, with some epigastric pain or discomfort, is all that is experienced.

The *physical examination* in cases of hæmatemesis is either negative or shows the various conditions of the stomach producing the bleeding, such as ulcer and cancer. In hæmorrhage from the stomach from whatever condition of the organ, there is frequently, however, some diffuse tenderness, which may not have been present before, as in cirrhosis of the liver, or which may be an aggravation of the previous tenderness, as in ulcer and catarrh; and in ulcer the localised tenderness may be situated in a larger area of slighter tenderness. Otherwise the stomach may be found dilated, the liver enlarged, a new growth present in the stomach or liver, or ascites may be discovered (see Diagnosis).

Melæna.—Bleeding from the stomach may be shown by hæmatemesis, and blood in the motions may be present at the same time. In severe hæmorrhage from the stomach, hæmatemesis is always present, but when the bleeding is slight and from a lesion near the pylorus, practically all the blood may pass into the duodenum and be voided as black blood in the motions. The causes of melæna are in part the same as those of hæmatemesis, *i.e.* when there is a large amount of blood present in the stomach some is passed in the motions, and thus it may occur when blood is swallowed and when it has been poured out into the stomach. It may also be due to hæmorrhage from the small intestine, in duodenal ulcer, from new growth, and from simple or from infective ulceration. In cases of ulceration of the large gut, whether infective (dysenteric), simple or malignant, melæna also occurs. In melæna produced by blood from the stomach or from the small intestine, the fæces are of a uniformly dark chocolate colour, differing greatly from the coal black colour of fæcal matter produced by iron salts, or the metallic black colour given by bismuth. No naked-eye diagnosis of melæna can be made if these drugs are being given; but when they are not being administered the colour of the stools is quite characteristic of melæna (Chapter V.). As regards symptoms these depend on the degree of hæmorrhage. If this is slow and continued for a long time (*i.e.* days) there is gradually increasing anæmia and weakness unaccounted for by any other condition present in the body. But this is not a very common occurrence in ulcer of the stomach. When melæna is present without hæmorrhage, and there are sudden symptoms of faintness, anæmia, and the other signs of internal hæmorrhage previously described, the bleeding is not from the stomach but usually from the duodenum or small intestine, as in duodenal ulcer (see Diagnosis of Ulcer, p. 433). The diagnosis of the cause of melæna in chronic cases lies practically between gastric and duodenal ulcer.

Diagnosis—Hæmatemesis.—In the diagnosis of hæmatemesis several points have to be considered which have been already indicated. The points to decide are:—

1. Is the stomach the seat of the hæmorrhage?
2. If the stomach is the seat of hæmorrhage, what lesion present: mechanical congestion, catarrh, ulcer, or carcinoma?

1. *Is the Stomach the Seat of Hæmorrhage?*—On referring to the list of causes of hæmatemesis unassociated with hæmorrhage into the stomach (p. 364), it will be seen that most of them present no difficulty in their recognition. In *epistaxis* the bleeding from the posterior part of the nose is usually accompanied by a flow of blood from the nostrils, and if there is no sign of blood anteriorly, an examination of the throat demonstrates the flow of blood posteriorly. If, as in some cases, the vomiting of the swallowed blood occurs some time after the epistaxis has ceased, the history of the nose-bleeding is usually clear, since hæmatemesis in these cases usually only occurs where there has been a profuse loss of blood from the nose. In fact in many cases of swallowed blood there is no hæmatemesis, because the gastric juice partially digests the blood, and the stomach expels it into the duodenum.

In the case of the rupture of a large artery (such as the aorta) into the upper alimentary tract, the bleeding is profuse and fatal. Some of the cases may not be seen during life, and are only cleared up by a post-mortem examination.

It is in cases of profuse hæmoptysis that difficulty arises in the diagnosis of bleeding from the stomach. The difficulty arises partly from the incomplete account the patient is able to give of either a previous or the present hæmorrhage, and partly from the fact that in the actual observation that a large quantity of blood has been brought up through the mouth, it is difficult to say whether the blood is vomited or not, especially as in cases of diseases of the lung, vomiting may occur at the time of hæmorrhage, or the blood in hæmoptysis may be swallowed or subsequently vomited.

Profuse hæmoptysis may arise—

1. From the rupture of an aortic aneurysm into a bronchus or trachea; this is rapidly fatal.
2. In pulmonary tuberculosis—
   - (*a*) In the early stage of the disease; initial hæmoptysis.
   - (*β*) In the course of the disease which is progressing.
   - (*γ*) From the rupture of an aneurysm of a branch of the pulmonary artery in a cavity; this is commonly fatal.

The practical question of diagnosis therefore lies between hæmoptysis in the various stages of pulmonary tuberculosis and hæmatemesis, and it is decided in the following ways: by the mode in which the blood is brought up, by the symptoms and physical signs present, and by an examination of the blood and other matters which are ejected.

The Mode in which the Blood is brought up.—In hæmatemesis there is one and only one bringing up of blood, this may be repeated once or twice, but at the end no more blood is brought up. In hæmoptysis, the mode of bringing up the blood is different. In the case of rupture of an aneurysm in a cavity, the bleeding is usually fatal; when recovery from the hæmorrhage takes place, the blood is continued to be brought up in small quantities mixed with sputum, and it is brought up by coughing. In initial hæmoptysis and the hæmoptysis of progressing pulmonary tuberculosis, there may be one large loss of blood at first, but the cases are rare, and are then not commonly due to tuberculosis, in which the patient does not for the rest of the day, or for the succeeding days cough up some blood mixed with expectoration. Close questioning of the patient will usually elicit this fact in cases of hæmoptysis. Another aid in the diagnosis consists in the following:—a patient may give the history of a large hæmorrhage, which is a doubtful hæmatemesis or hæmoptysis, but inquiry will usually bring out the fact that there have been subsequent slight hæmorrhages, in which the blood was coughed up with expectoration, leading to the diagnosis of the first hæmorrhage being a hæmoptysis and not a hæmatemesis. The continued coughing up of blood after the initial hæmorrhage, which is so characteristic a feature of hæmoptysis, is due to the fact that in pulmonary tuberculosis slight hæmorrhage (capillary) continues after the first, and also to the peculiarity of structure of the lung, which retains some of the exuded blood, which is subsequently coughed up of a dark colour.

The *symptoms and physical signs* present may indicate the source of the blood. In bleeding from the stomach there is the previous history and the actual presence of the stomach symptoms characteristic of ulcer. In some cases, however, the loss of blood is apparently the first symptom in ulcer, but this is not really the case, since a close questioning brings out a history of symptoms of indigestion of food or of localised gastric pain after food. Similarly in hæmoptysis there are the previous symptoms of lung disease, cough and expectoration, while an examination of the chest will reveal the signs of disease of the lungs. In the early hæmoptysis of pulmonary tuberculosis, physical signs may be so doubtful that no conclusion can be drawn from them, and there are cases where a single hæmoptysis has occurred and no physical signs have afterwards been discovered. In these cases the decision of the existence of hæmoptysis must rest on the other grounds described and on the presence or absence of fever and night sweats. In cases of hæmatemesis due to portal obstruction, the existence of ascites, of pain and tenderness in the liver region, of slight enlargement of the liver, and the proof of the alcoholic habits of the patient, are aids in the diagnosis.

The *examination of the ejected blood and other matters* is of great importance. Following a profuse hæmoptysis, there may be sputum, stained with blood and containing pigment cells and tubercle bacilli, and the mixture is alkaline; with a hæmatemesis, the blood is usually mixed with the contents of the stomach, and since bleeding from the stomach usually occurs after a meal, the vomit is acid, containing hydrochloric acid and the remains of the meal. A naked eye, a microscopical and a chemical examination of the ejected blood reveals this composition of the fluid and aids the diagnosis (Chapter V. p. 123).

2. *If the Stomach is the Seat of the Hæmorrhage, what Lesion is present?*—The decision of this point rests almost solely on the recognition of the symptoms peculiar to the lesion of the stomach. Where an aneurysm ruptures into the stomach death follows, and the disease may have been diagnosed during life by an examination of the abdomen. In the case of catarrh, hæmatemesis is an infrequent symptom, and only slight in amount; in ulcer a similar slight hæmatemesis may occur, and the diagnosis between the two conditions is made by a reference to the symptoms peculiar to each (see Ulcer, p. 433). In a profuse hæmatemesis due to a stomach lesion, the diagnosis lies between ulcer, mechanical congestion, and carcinoma. In carcinoma, as a rule, the diagnosis is not difficult, since the coffee-grounds vomiting is usually present with the signs characteristic of the disease, viz. persistent pain, the presence of a tumour, dilatation of the organ and bacterial fermentation, with the general symptoms of the disease. Between the hæmatemesis of old ulcer with dilated stomach and that of carcinoma, the diagnosis is often extremely difficult, and depends almost solely on the history of the case.

The diagnosis between the hæmatemesis of portal obstruction and that of ulcer not infrequently presents difficulties at the outset. The symptoms and signs peculiar to each disease must be examined. Thus in cirrhosis of the liver (the commonest cause of portal obstruction), the patient is usually a male, addicted to alcohol, and he may show an enlarged tender liver, and ascites; while in ulcer the patient is usually a young female with the characteristic localised pain after food. But cases sometimes present great difficulty, because in cirrhosis of the liver, there is frequently indigestion of food (irritation or catarrh) and vomiting, while in ulcer the localised pain after food may be absent at the time of examination, and the patient may be a man of middle age. In the diagnosis of the occurrence of bleeding from the stomach and of the particular lesion producing it, no one fact is to be relied upon but all must be considered together.

Prognosis.—The prognosis of bleeding from the stomach depends on the amount of blood lost and the nature and stage of the disease causing it.

Thus in catarrh, the small amount of blood lost, although an important sign, does not affect the patient profoundly. In cancer and ulcer, on the other hand, hæmatemesis may be a serious sign. In cancer, the slow loss of blood as shown by a subsequent hæmatemesis is one of the factors which aid the downward progress of the patient, and it may be the final event before death.

It is in ulcer where the question of the prognosis is of importance. Bleeding from the stomach is usually shown in these cases by hæmatemesis. Slight hæmatemesis in the course of a case of ulcer, although in itself producing but slight effect on the patient, is an indication for treatment directed to prevent a further bleeding.

In profuse hæmatemesis, the immediate prognosis is as a rule good, even though the hæmorrhage may be severe; such patients rarely succumb immediately to it. But in determining the prognosis, several conditions must be considered besides the amount of blood lost, viz. the general condition of the patient, and the special condition of the stomach. In cases, for example, of old ulcer, when the patient is greatly wasted, has been worn out with pain, and has been capable of taking very little food, the occurrence of hæmatemesis is a serious symptom and may lead to death. These debilitated patients cannot withstand the loss of blood.

On the other hand, in the more usual forms of hæmatemesis, such as occur in cases of ulcer in young women, the loss of blood has a profound but not a fatal effect on the patient. It may be weeks or months before a complete recovery takes place, but with care the patient does get well. The recovery from the hæmorrhage is in great part due to the measures of treatment adopted.

In the hæmatemesis of portal obstruction, the immediate prognosis is also good, although it may be one of the factors aiding the final dissolution of the patient. The condition of the stomach in all cases has an influence on the hæmatemesis, which is always a more serious sign if the organ is dilated and unable to contract than when the muscular power is but little weakened.

Treatment.—The treatment to be adopted in cases of bleeding from the stomach is the same as the treatment of profuse hæmorrhage from other parts. The essential of such treatment is rest—rest to the body, rest to the stomach and, what is not quite the same thing to all minds as rest, the abstention from all active treatment.

Immediate Treatment.—The patient is to be kept in bed and is to remain quite quiet in the recumbent position. If the bowels have not been open, as soon as the first effects of the hæmorrhage are past, an enema must be given to open them. The visits of friends are to be strictly prohibited for a time, only the attendants being allowed to be present. All food by the mouth is to be stopped in cases of ulcer, only a little ice being allowed to relieve thirst, and rectal feeding is to be adopted (p. 343).

When there is syncope or a tendency to syncope after the hæmorrhage, this may be treated either by the application of a cold damp cloth to the temples or by allowing the patient to inhale the vapour of ammonia or of strong smelling salts, and by giving a rectal injection of half an ounce of brandy with half an ounce or an ounce of warm water. If the syncope is well marked 5 or 10 minims of ether may be injected subcutaneously. Beyond this, however, no vigorous measures for restoring the patient are to be adopted. The administration of stimulant drugs by the mouth is not to be recommended, and anything beyond gentle rubbing of the patient's hands and feet is to be avoided. The feet and legs are to be kept in a raised position by pillows. The recovery is usually rapid and is indicated by returning consciousness (if this has been lost) and by the condition of the pulse, which becomes less frequent and more bounding.

In all cases of ulcer it has to be considered whether means are to be taken to stop the hæmorrhage or prevent its recurrence, or whether transfusion is to be performed.

The rest of body and rest to the stomach described above, with the sucking of ice, are often the only means necessary to employ to prevent a recurrence of the hæmorrhage. But in many cases it is necessary to prescribe astringents to prevent a recurrence of the hæmorrhage, which would be a serious event for the patient, and in those cases of ulcer where there is recurrent melæna without hæmatemesis astringents are useful.

The astringents to be given are three in number:—

Acidum tannicum, in doses of 2 or 5 grains in water or pill.
Acidum gallicum, in doses of 2 or 5 grains in water or pill.
Plumbi acetas, in doses of 1 or 2 grains in pill.

The administration of small doses frequently, every one, two, or three hours, according to the emergency of the case, is the best method of giving the drugs. Large doses are apt to cause vomiting, which would be highly injurious to the patient.

Transfusion.—In those cases of profuse hæmatemesis occurring in the course of ulcer and in a greatly debilitated person, transfusion has always appeared to be worthy of a trial. It is to be remembered that ulcer of the stomach in the great majority of cases is not a fatal disease, and that where such an event as profuse hæmorrhage occurs, and the patient is likely to die from the loss of blood, it is evident that transfusion may save the patient's life, as it undoubtedly does in some cases of post-partum hæmorrhage. Such cases fortunately do not often occur in ulcer.

The best liquid to use for transfusion is a solution of common salt (1 drachm to the pint), and the amount to be injected is from one to two pints—two pints if possible. The solution is now prepared in convenient sterile tubes, each of which contains 2 drachms of common salt dissolved in a little water; the point of the tube is broken off, and the liquid poured into water (tap-water) which has been previously boiled and cooled to about 105° F., the proper temperature for the transfusion liquid.

Soon after the hæmorrhage, other symptoms may arise—restlessness and excitement. These occur after great losses of blood, and are to be treated by the cautious administration of sedatives. One of the best of these is a hypodermic injection of morphine, 2 minims of the injectio morphinæ hypodermica (B.P.) being given. This injection is to be repeated only in rare instances. In other cases the excitement and restlessness is well controlled by an enema of chloral hydrate (10 grains) and potassium bromide (30 grains) in 2 ounces of warm water.

Excessive vomiting and retching occurring during a hæmatemesis or afterwards is likely to lead to a recurrence of the hæmorrhage. A hypodermic injection of morphine is the best method of treating this condition.

Practically all the remarks made regarding the treatment of hæmorrhage from the stomach refer to cases of ulcer. In hæmatemesis from portal obstruction, the loss of blood is frequently not so serious a sign as in ulcer, and is in many cases a relief to the venous congestion and alleviates the symptoms of the patient.

Subsequent Treatment of Cases of Bleeding in Ulcer of the Stomach.—The objects in view are to give the stomach a complete rest, so as to prevent the recurrence of hæmorrhage, and to enable the ulcer to heal; also to treat the anæmia produced by the bleeding.

It is in such cases that prolonged rectal feeding is of such great benefit (Chapter XI. p. 343); and this is undoubtedly the treatment to be adopted if the patient can bear it. By it complete rest is given to the stomach, while the strength of the patient is kept up.

For the treatment of the anæmia, iron is to be administered. It may be given in the nutrient enemata in small doses of perchloride or sulphate of iron, or it may be cautiously administered by the mouth, when food is again taken into the stomach. The further treatment of the case is that of ulcer (see p. 437).

CHAPTER XIV.

DILATATION OF THE STOMACH (DILATATIO VENTRICULI, GASTRECTASIA). CIRRHOSIS VENTRICULI.

DILATATION OF THE STOMACH.

DILATATION of the stomach is a symptom which, from its frequent occurrence and special treatment, must be considered separately. The essential cause of dilatation is a weakness of the muscular coat of the organ, produced by various causes, but in addition to this there is a distending force, which consists of the food taken and the products of bacterial decomposition of the food. These two causes act together. In some cases the muscular weakness is slight at first, but the distending force is great, and *vice versâ*.

Causes of Muscular Weakness.—These have already been considered in the preceding pages, but they may be again summarised.

(*a*) *The Stomach Muscles may have more work to do than they can accomplish.*—This may arise either from an excess of food being taken at one time, or from an obstruction to the passage of food into the duodenum, or from duodenal obstruction. Up to a certain point, hypertrophy occurs to accomplish the extra work thrown on the muscles; but this is soon followed by diminished work, as shown in increased dilatation. As in the heart, although not to so great an extent, dilatation precedes hypertrophy; dilatation being the condition which hypertrophy counteracts. Hypertrophy of a muscle is, however, dependent on the state of nutrition of the muscle, so that it is most commonly seen in young muscles, and where the distending force is not great. In the case of the urinary bladder, for example, when there is a stricture of the urethra impeding the emptying of the organ, this tends to dilate but also hypertrophies to overcome this dilatation and the obstruction in front. The degree to which hypertrophy exceeds dilatation depends on many causes, one of which is the age of the patient; for, speaking generally, urethral stricture in a young man tends to produce hypertrophy of the bladder, while prostatic obstruction in an old man tends to produce dilatation.

In the case of the stomach, dilatation is much more frequently seen than hypertrophy, owing chiefly to the fact that the condition producing it is prolonged, and that the distending force is of varying intensity, at some times very great, at others less.

Pyloric stenosis, whether due to cancer or to fibroid contraction, leads to hypertrophy of the muscle, but also to dilatation, so that in one case there may be chiefly hypertrophy, in another hypertrophy and dilatation, and in a third dilatation without hypertrophy. The conditions found are, indeed, like those observed in the cases of mechanical obstruction to the passage of the urine just mentioned. In pyloric stenosis due to cancer, cases are met with in which there is no dilatation, in which the stomach is indeed contracted (see Cancer, p. 467). These are classed as cirrhosis ventriculi, and the condition in these is that there is hypertrophy of the muscular coat, and in addition, infiltration of the walls of the stomach, partly fibroid and partly cancerous. The result is a stiff and unyielding wall which cannot dilate.

(β) *There may be a Primary Weakness of the Muscular Wall.*—Examples of primary weakness of the muscular coat are met with in cases of gastric insufficiency, where it is due to a general condition of the body, usually anæmia. In all cases of gastritis this primary weakness of the muscle is present and is due to the inflammatory condition. Lastly, the nervous system may be the chief cause of the primary muscular weakness. There is no doubt, clinically, that some cases of dilatation are mainly due to the effect of the nervous system; this may occur in cases of gastric insufficiency, but it is more obvious in cases of nervous dyspepsia, and in hysteria and neurasthenia. It is probable also that in those cases described as acute dilatation of the stomach, the primary effect is a nervous one, acting perhaps in some slight chronic condition of the stomach or in a debilitated patient.

A primary degeneration of the muscle fibres leading to muscular weakness must be considered as rare in the production of dilatation. It may occur in phosphorus poisoning or in acute inflammation, but is usually a sequel of dilatation and not its cause.

The Distending Force in Dilatation.—This is the food which is either taken in too great a bulk to be manipulated by the organ, or is delayed in the organ either by simple muscular weakness or by pyloric obstruction. The greatest distending force is, however, the accumulation of gas, and this continuing for long periods tends to produce dilatation of the organ. Especially is this so when there is bacterial fermentation of the food, and cases of dilated stomach are precisely those in which this most commonly occurs. The effect of unsuitable food in causing and increasing dilatation is well seen in neglected cases of gastric insufficiency and gastric catarrh, such cases being aggravated and prolonged by the eating of food which the stomach is unable to digest and manipulate. By ordering a suitable diet, or by the withdrawal of all food taken by the mouth, the dilatation of the stomach is observed to decrease greatly, often in the course of two or three days.

For practical purposes, dilatation of the stomach is most conveniently classified under two headings, *obstructive and non-obstructive.*

Obstructive Dilatation—

1. Occurring in stenosis of the pylorus, caused by a cancerous new growth or by fibroid contraction.

2. Occurring after the cicatrisation of a large chronic ulcer of the pyloric region.

3. Following pressure on the duodenum by a new growth in the peritoneum, rarely in the pancreas, or following stricture of the duodenum caused by the cicatrisation of an ulcer (rare).

4. Traction on the pylorus causing a partial stenosis. This occurs from the adhesions formed in chronic peritonitis.

5. Traction on the cardiac end of the stomach by surrounding adhesions which may result from a severe and chronic left-sided pleurisy.

Non-obstructive Dilatation—

1. Occurring as a sequel of gastric irritation. In these cases the dilatation may at first be well-marked only during the digestion of a meal, the stomach ultimately recovering.

2. More commonly occurring in gastric insufficiency, in some cases of which the muscular weakness is more marked than the deficiency in gastric juice.

4. Still more important is the dilatation which is the result of subacute catarrh and of long continued chronic catarrh. Whereas in gastric irritation and gastric insufficiency the dilatation is only moderate in degree, in catarrh it may range from a moderate to a very great degree.

The most common causes of great dilatation of the stomach are cancer of the pylorus and catarrh: of moderate dilatation, the commonest cause is gastric insufficiency.

General Pathological Condition present in the Dilated Stomach.—Although there are various pathological causes of dilatation of the stomach, yet the condition is associated with certain morbid changes which are always to be borne in mind. In the first place, the process of digestion is very imperfect. This is due not only to the great motor weakness, but to the deficiency in the secretion of the gastric juice, and to the cessation of absorption by the mucous membrane. The secretion of pepsin and of hydrochloric acid does not completely cease, but the hydrochloric acid being deficient, the chemical processes cease after a short period of digestion, and in the liquid removed from the stomach no free hydrochloric acid may be found, showing that it has disappeared. Again, the great muscular weakness leads to delay of food in the organ, and although most of the solid food may be more or less transformed, yet the resulting liquid is not absorbed, and contains peptones and carbohydrates in solution. The absence of hydrochloric acid at one or other period of digestion, and the delay of food in the organ, lead to the commonest result of dilatation, viz. bacterial fermentation of the food.

In cases of dilatation of the stomach, where there is no stenosis of the pylorus, this becomes patent and allows the contents of the duodenum to enter the stomach; thus both pancreatic juice and bile may enter the stomach in this way. The presence of bile, therefore, in the stomach contents is of some importance in the diagnosis of these conditions, as excluding stenosis of the pylorus. Gases may also pass from the small intestine into the stomach, and it is probably in this way that marsh gas and sulphuretted hydrogen enter the organ, and are eructated (see Gases of the Stomach, p. 97).

Symptoms.—The symptoms of dilatation are associated with and often obscured by those of the disease producing it, but there are certain special signs which are due to the dilatation alone, and these will now be considered, chiefly as they occur in cases of great dilatation.

As a rule the symptoms have no direct reference to the ingestion of food, although there are exceptions to this, as in the cases occurring in the course of catarrh and of ulcer. The symptoms may come on four, five, or six hours after a meal, or they may have no reference to the meal at all, the most pronounced symptoms occurring at intervals of twenty-four hours, or even two or three days.

These symptoms are epigastric distress and pain, vomiting, gaseous eructations, and reflex symptoms, either pain or those referable to the heart and respiration.

The *epigastric distress and pain* precede the vomiting. They are not localised, but diffused all over the stomach region. In cases where the dilated stomach sinks in the abdomen, the distress is more in the umbilical than in the epigastric region. It is sometimes very severe, being accompanied by a hot burning sensation, and begins as a slight uneasiness which increases until vomiting relieves. So closely is this symptom associated with the character of the stomach contents, that patients after a time excite vomiting by putting their fingers down the throat. The epigastric pain is due in most cases to the irritating products of bacterial fermentation, but in others, *e.g.* catarrh, it is increased by the inflammatory condition of the organ. Nausea, although rare, is sometimes present.

Vomiting.—The typical vomiting occurring in dilatation of the stomach consists in the bringing up of large quantities of very acid fluid at long intervals. The fluid accumulates in the dilated stomach for twenty-four or thirty-six hours or longer until it is thrown up, and the high acidity is due to the presence of the organic acids produced by bacterial fermentation When brought up, this fluid is exceedingly irritating, producing a hot, burning sensation in the chest, pharynx, and mouth, and setting the teeth on edge.

The amount of fluid vomited varies; it may be two, four, or even six pints, and it possesses the characteristics already described (p. 93). It may contain bile, and blood of the colour of "coffee-grounds." Bile is not constantly present unless there is duodenal obstruction beyond the entrance of the common bile duct.

Gaseous eructations (flatulence) in large quantities are a feature of dilated stomach. The gas is one of the products of bacterial fermentation, and is of varying composition (p. 97). The chief component is carbonic acid (CO_2). Flatulence is very severe during the accumulation of the liquid in the stomach, but in some cases after vomiting, a large production of gas takes place, blowing out the stomach, and causing great distress.

The other symptoms in dilatation of the stomach are partly referred pain of the character already described as occurring in functional disorders of digestion, and partly those symptoms which are due to the presence of a large tumour in the abdomen, which affects the heart and respiration. These effects are the more marked, the more acute or sudden is the dilatation of the stomach. Thus in those cases which have been called acute dilatation, there is great depression of the body generally, and great embarrassment of respiration and of the circulation of the blood; the former being shown in dyspnœa and the latter in a rapid and an irregular pulse of low tension. The stomach is at this time full of liquid and gas, and unless it is emptied rapidly, death may ensue (Hilton Fagge). In chronic dilatation such severe symptoms do not occur, but an effect on the respiration and circulation is frequently observed. Before the vomiting, *i.e.* when the stomach is full of liquid, dyspnœa is often present; and may be ascribed to the enlarged and heavy organ interfering mechanically with the action of the diaphragm and the expansion of the lungs. Rapidity of the pulse, or irregularity, is also sometimes present, palpitation being a frequent symptom complained of by these patients. This must be mainly a reflex effect of the dilated organ. Relief is given to these symptoms by removal of the stomach contents.

Effect on General Nutrition.—In chronic cases of dilatation, especially when there is bacterial fermentation, general wasting is frequently observed, and there may be great emaciation. This is seen in cases of simple (non-obstructive) dilatation as well as in the obstructive form. In simple dilatation, it is due chiefly to the greatly diminished amount of food which is absorbed; and this is partly due to the fact that the patient takes little food owing to the distress it causes, but also to the fact that this small amount of food is imperfectly digested, a portion of it undergoes bacterial fermentation, and a not inconsiderable portion is vomited. In cases of obstructive dilatation these conditions are more marked; hence the wasting is greater, but it is greatly aided in this case by the presence of a malignant growth not only in the stomach itself, but in the liver and peritoneum.

The great improvement in general nutrition which follows the treatment of dilated stomach, even in cancer, demonstrates that the wasting observed is largely due to the conditions present in dilatation of the organ.

Effect on the Intestines.—As a rule the bowels are obstinately constipated in dilatation of the stomach, but they may become loose and offensive when there is bacterial decomposition along the intestinal tract. There may be associated dilatation of the intestines, usually of the large gut, which is greatly distended with gas; in some instances the small gut is similarly distended, but the condition is a passing one.

Effect on the Urine.—A small quantity of urine is passed, of high specific gravity, and containing often an excess of phosphates and a large quantity of ethereal hydrogen sulphates (p. 119).

Physical Examination.—The physical signs of dilated stomach are most evident when the stomach contains a large amount of liquid, or is distended with gas. In some cases it is advisable for the purposes of diagnosis to distend the organ artificially with gas. This is readily done by making the patient drink 15 to 30 grains of citric or tartaric acid dissolved in half a tumbler of water, and immediately afterwards 15 to 30 grains of bicarbonate of sodium dissolved in a similar amount of water. The carbonic acid evolved in the stomach distends the organ, which is thus easily examined.

Inspection.—In moderate degrees of dilatation there is a prominence in the lower epigastric and upper umbilical regions, rounded and ill-defined; but when the dilatation is great, the stomach sinks somewhat and the upper edge of the prominence is sometimes well-defined by a groove extending from just above the umbilicus upwards and towards the left hypochondrium. This groove marks the lesser curvature. The lower limit of the swelling is often not so well-defined, gradually fading off into the hypogastrium, but in most cases it is also marked by a groove which is situated transversely below the umbilicus, and midway between this and the pubes. This marks the greater curvature. It is not a straight transverse groove, but towards the left lumbar region turns upwards to the left hypochondrium.

When the stomach is full of liquid or gas, or is irrritable as in cases of catarrh, vermicular action may be seen as a wavy motion along the lines of the lesser and greater curvature. As a rule when the stomach is empty, and when there is a greatly dilated flaccid stomach, no vermicular action is seen.

Palpation confirms inspection in so far that the greater curvature is frequently to be felt as an ill-defined round edge below the umbilicus, becoming indistinct as it is traced to the right and left. The sense of resistance varies over the dilated organ, according to the degree of distension. When it is full of gas there is increased resistance all over the organ, *i.e.* in the lower epigastric and umbilical regions; and the impression may be given of palpating a hard ball. In other cases there is no great increase of resistance, the abdomen being enlarged, but flaccid everywhere.

On manipulating the stomach in cases of dilatation in subacute catarrh, it frequently can be made to contract, the lower edge passing from below the umbilicus to the level of the umbilicus or even above it.

In some cases the cæcum and colon, when distended, can be readily felt by palpation, the cæcum being very evident; while to the right of the stomach is the distended ascending colon and part of the transverse, and below the stomach the remaining part of the transverse colon.

Splashing is a sign to be obtained by palpation of the dilated stomach when it contains liquid. It is produced by a sudden jerk of the hand placed flat over the distended organ. It is practically diagnostic of the condition. It may be obtained in moderate cases of dilatation when the stomach is fasting. Pulsation in the epigastrium is not infrequently present in dilatation of the stomach; it is not significant of the condition, and when the organ is full of liquid in cases of moderate dilatation, a systolic epigastric thrill may be felt. It is of no great importance as a sign.

Percussion.—In not a few cases the area of the stomach can be mapped out in the abdomen by means of percussion. The area so defined is not a reliable indication of the degree of dilatation of the organ, since it is found that this is capable of greater distension than diagnosed, when it is inflated with gas.

Percussion over a dilated stomach usually gives a tympanitic note, the characters of which it is very difficult to define in words; it is short, high-pitched, and somewhat musical in quality. It is sometimes called a "stomach" note, but it is often impossible to distinguish it from the percussion note over a distended colon. Where percussion is of value in the recognition of dilated stomach is in those cases of moderate dilatation, or of dilatation when the organ does not sink in the abdomen, and where the cardiac extremity pushes up the diaphragm. In this case the stomach note is obtained in the lower part of the left axillary region, forwards to a varying extent, backwards as far as the posterior axillary line, and upwards as far as the fourth rib. These signs are obtained in cases of continued moderate dilatation and in those cases of gastric insufficiency where a temporary dilatation or a temporary increase of the dilatation occurs an hour or two after a meal.

In cases where the cardiac extremity is adherent to the diaphragm, and the organ is dragged up by chronic disease of the left lung and pleura, as in some cases of pulmonary tuberculosis, the position of the organ may be determined by the permanence of the stomach note in the axillary region or just in front of it. It is usually only a small area over which this permanent stomach note is obtained.

Auscultation is of but little value in the recognition of dilated stomach. The splashing of fluid may be heard, and if the patient be given some water (2 or 3 ounces) to drink, the liquid may be heard to fall into the dilated stomach with a characteristic sound. This sign is not obtained in the normal organ, but is obtained in cases of cirrhosis of the stomach as well as in dilatation.

Other Aids to Examination of the Stomach.—The presence of dilated stomach may be detected by the passage of a moderately firm sound, the end of which may be felt at the greater curvature, at the level of the umbilicus or below. The degree of dilatation as well as its actual presence may be seen by the amount of liquid which can be poured into the stomach through the tube and regained. The capacity of the normal stomach is from 800 to 1200 cc. (from 1½ to 2½ pints). In cases of dilated stomach three or four pints of liquid may be retained, and regained by the aspirator or siphon.

Diagnosis.—The diagnosis of dilatation of the stomach comes under two headings :—

1. As to its existence.
2. As to its causation.

1. The *existence* of dilatation is to be determined by the symptoms and method of examination just discussed. The symptoms which point to dilatation are the character of the vomiting and of the vomited matters. The periodic vomiting of large, often enormous, quantities of chiefly fluid matter with a few flocculi of undigested food, and containing a large percentage of organic acids, as well as yeast and sarcina is diagnostic of dilatation. The signs of bacterial fermentation

may be wanting at the period of first examination; but the quantity of the liquid vomited serves as a sure guide to diagnosis.

The patient may be seen when the vomiting has lessened or even ceased; but the history of the vomiting is usually, however, quite clear, and a physical examination will render the diagnosis easy in most cases.

In moderate cases of dilatation percussion is a great aid to diagnosis, especially in the extension of the stomach note into the axillary region. This sign may be supplemented by artificial inflation of the stomach in the manner described.

In cases of great dilatation the physical signs observed may render the diagnosis obvious, but they are supplemented with advantage by artificial inflation and by washing out the organ, an examination of the washings for organic acids, bacteria, and sarcina being made.

2. The *diagnosis of the causation* of dilatation of the stomach often presents great difficulties.

When it occurs after long continued gastric irritation, or in gastric insufficiency and catarrh, the diagnosis is to be determined on the lines previously laid down (p. 262). These are usually cases of moderate dilatation. In cases of great dilatation the diagnosis rests between the presence of *malignant* or *non-malignant* disease; and the question must always arise in one's mind, Is the disease cancer or not? There are cases where the diagnosis is comparatively easy, where there is a tumour of the pylorus, and the well-marked symptoms of malignant disease, wasting and anæmia. But in the absence of a tumour the diagnosis is not so simple. In cases of subacute catarrh, as a rule, there is no difficulty; the history of the illness, and the presence of mucus in the vomit and the stools making the case clear. When, however, there is great dilatation of the organ without catarrh and no tumour is present, the case may be one of malignant disease or of simple dilatation. In such cases the various facts hereafter discussed as characteristic of malignant disease must be taken into consideration (p. 473). In many cases no diagnosis is possible until some time has elapsed.

Prognosis.—The prognosis in dilatation depends on the cause of the condition. In cases of moderate dilatation the prognosis is the same as the condition producing it—gastric insufficiency or catarrh. In cases of dilatation in subacute catarrh, the prognosis is as a rule good; with prolonged treatment such patients recover to a very great extent. In great dilatation, whether simple or malignant, the condition is greatly improved by treatment. In simple dilatation, although complete recovery of stomach function may not occur, the patient with care may recover sufficiently to lead a useful existence, although death does occur in some cases from exhaustion. In malignant disease no recovery is possible, but patients improve greatly under appropriate treatment, *i.e.* treatment applied to the relief of the dilatation.

Treatment.—The treatment of moderate dilatation of the stomach is the treatment of gastric insufficiency and of catarrh, but in these cases, besides the special treatment of the disorders, medicinally and by diet, general hygienic treatment and massage (Chapter. XII.) are of great value. Washing out the stomach is in these cases an emergency, and is not to be recommended for continued use.

In great dilatation in subacute catarrh washing out the stomach is as a rule contraindicated, and the patient is to be treated as previously directed (p. 249).

When there is great dilatation with bacterial fermentation, a daily washing out of the organ is the best treatment (Chapter XII.), and preferably by an antiseptic solution. Under this treatment the patient greatly improves, and the dilatation becomes less, even when there is pyloric obstruction. Antifermentative remedies may also be given, and they are imperative in those cases where the passage of the stomach tube causes great distress. The daily washing out with the administration of antifermentatives cures the bacterial fermentation; and the great distress of the patient is thus relieved.

Massage, both local and general, and electricity are of some service, but only in combination with the above treatment.

Diet.—See Chapter XI. p. 347.

For a further consideration of dilatation caused by fibroid stricture of the pylorus, see p. 441, and of that caused by cancer, p. 467.

CIRRHOSIS VENTRICULI (PLASTIS LINITIS, BRINTON).

Definition.—A chronic inflammation of the walls of the stomach, associated with hypertrophy of the muscular coat, usually involving the whole organ and producing contraction.

The *etiology* of this affection is somewhat obscure. It occurs usually in men and in youth or middle age; but women are not exempt. It may be primary, or secondary to surrounding inflammation. According to Brinton, many of the subjects of cirrhosis ventriculi have been "either drunkards or dram-drinkers."

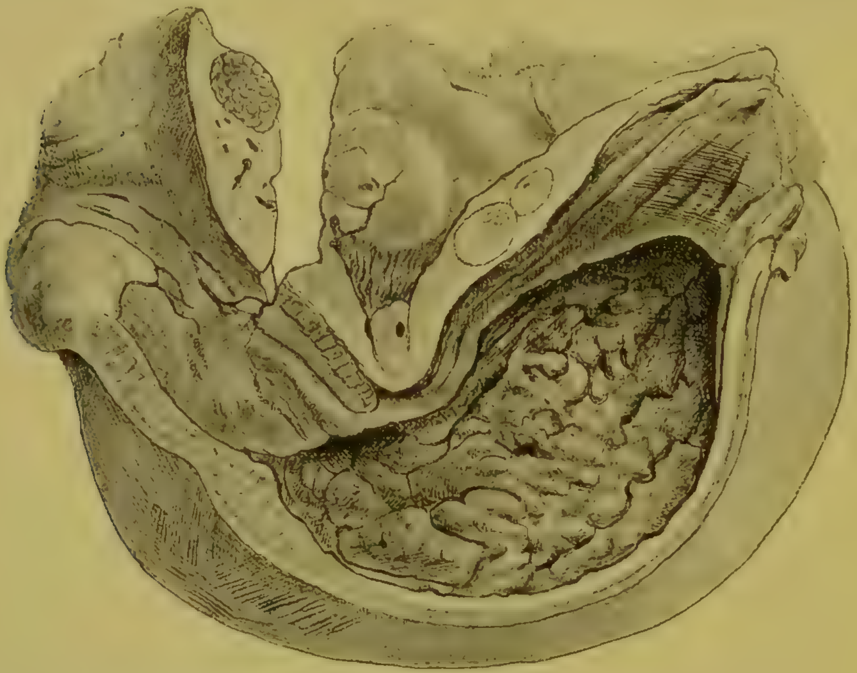

FIG. 33.—Cirrhosis ventriculi. $\frac{2}{3}$ natural size. The stomach is shaped like a leathern bottle. The cardia is to the right and the pylorus to the left. The walls of the organ are greatly thickened, especially near the pylorus : the thickening is due to hypertrophy of the muscle, to fibrosis and to the presence of nodules of sarcoma. Some enlarged and sarcomatous glands are seen above the lesser curvature, and there is great thickening round the duodenum. The œsophagus is normal. From a case of sarcoma of the retro-peritoneal glands in a young woman.

Morbid Anatomy and Pathology.—As the result of chronic catarrh a cirrhosis of the mucous membrane was described (Chapter VIII.), but this is a different condition to what is termed cirrhosis ventriculi, inasmuch as in chronic catarrh the fibroid change is limited to the mucous membrane and does not involve the other coats of the organ. The change in cirrhosis of the

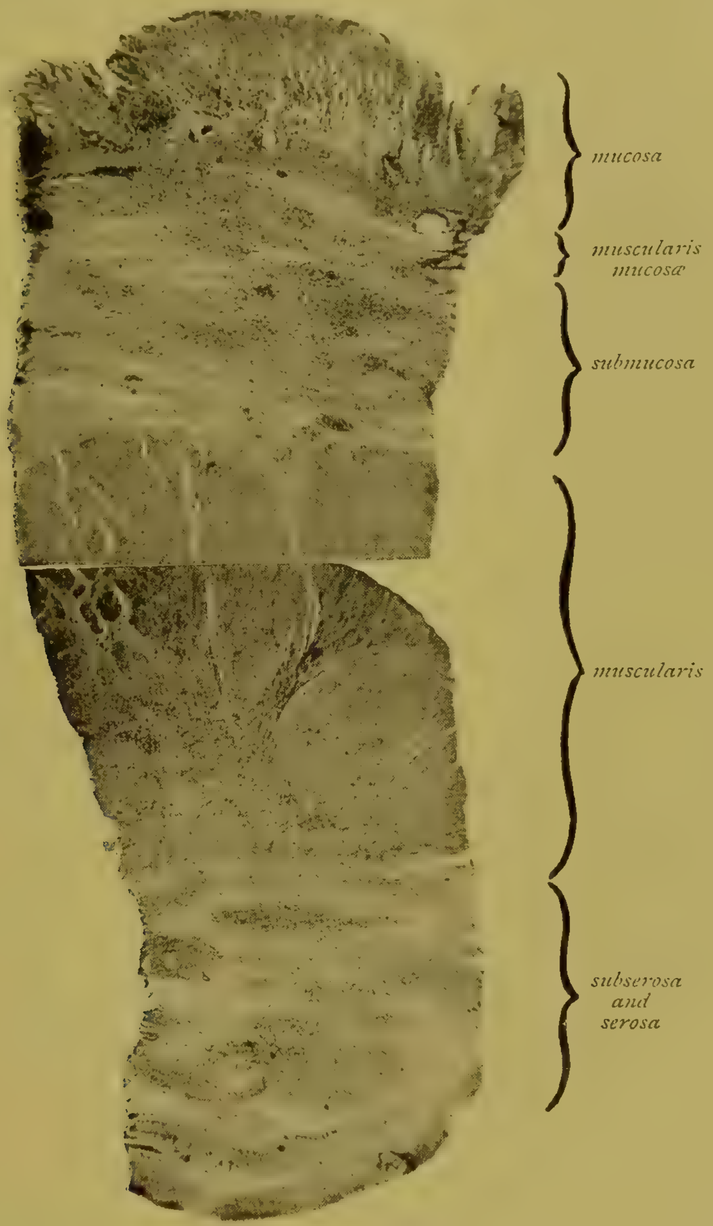

FIG. 34.—Microscopical section of the mid-region of the stomach represented in Fig. 33. From a photograph, × 18. The great thickening of the wall is seen to be due to three changes: (1) the great increase of fibrous tissue in the submucous and subserous coats; (2) the hypertrophy of the muscular coat and of the muscularis mucosæ; (3) the presence of nodules of sarcoma in the subserous and submucous coats, and to a less extent in the mucous. These are represented by the darker shadings in the figure. From a preparation hardened in alcohol and stained with logwood.

organ will be evident on looking at the accompanying figure (Fig. 33). The organ is pale, almost bloodless, contracted, stiff, with walls greatly thickened throughout, and on being opened does not collapse like an ordinary stomach, but remains rigid. It has lost its normal shape, the cardiac pouch having nearly disappeared, so that it approaches to the tubular form of a simple stomach, or, as is sometimes said, it is like a leathern bottle. The mucous membrane to the naked eye is thrown into longitudinal folds, and is bunched up in parts owing to the fact that it is less affected than the other coats of the organ by the fibroid change. The capacity of the stomach is greatly reduced; it may be as little as 4 ounces in extreme cases.

The microscopical examination, which is seen in the accompanying figure (Fig. 34), shows the nature of the change in the stomach walls.

It is noticed that the subserous connective tissue and the connective tissue of the submucous coat are greatly increased, causing part of the great thickening of the stomach wall. The vessels in the submucous coat are thickened, their fibrous coat being increased and the lumen greatly diminished.

The muscular coats are greatly hypertrophied. The muscularis mucosæ is three or four times its normal thickness. The outer muscular coating is also greatly hypertrophied, and the connective tissue and septa are increased in thickness. It is noticeable that five-sixths of this hypertrophy is due to the increase of the circular muscle fibres, the outer longitudinal layer being but slightly affected.

The mucous membrane is the coat least affected. It shows but little fibrosis; it is still covered with epithelium, and the gland cells present in many parts a normal appearance.

This description serves as a type of all cases of cirrhosis. The disease, however, varies in degree as regards the amount of fibrosis and the extent of hypertrophy of the muscular coat.

The causes of such a chronic inflammation with hypertrophy are difficult to determine. This is especially so in the primary cases in which there is no other disease present. The inflammatory process in this case may be supposed to be initiated by some irritant taken into the stomach, which produces an acute inflammation affecting all the coats and becoming chronic. Such acute diffuse inflammations of the stomach occur in cases of poisoning, and occasionally in the course of an infective disease. The inflammation may be kept up by the food which is taken. This is only, however, a theoretical explanation of these primary cases; but little is really known of their causation.[1] The hypertrophy which occurs in such cases is explained by the increasing rigidity of the stomach walls, which causes a mechanical difficulty in the expulsion of the contents.

A second class of cases of cirrhosis ventriculi occurs in which the explanation of the occurrence of the condition is evident. Thus it is found in some cases of chronic peritoneal inflammation of the upper part of the abdomen, affecting the liver, stomach, and spleen. This chronic peritonitis may be either simple or malignant. In the case described above, the origin of the inflammatory change in the stomach was obvious from the thickening of the peritoneum by fibrous tissue and by nodules of sarcoma, especially round the stomach and duodenum: this had caused a narrowing of the duodenum, which in part accounted for the hypertrophy of the muscularis of the stomach; only in part, however, since the rigidity of the stomach wall would also cause hypertrophy. Although in this case there were a few nodules of sarcoma in the stomach wall, which had evidently spread from the peritoneum, the condition is different from the diffuse form of carcinoma ventriculi, in which there is cancerous infiltration spreading through the coats of the stomach from the pylorus. In some cases of cancer of the pylorus there is a diffuse fibrosis with hypertrophy of the stomach (see Cancer, Chapter XVI.).

The chronic inflammation in the upper part of the abdomen may be non-malignant, due to an old peritonitis (usually tubercular), or to inflammation set up by a benign tumour (hydatid) of the liver.

Symptoms.—Cirrhosis ventriculi has been frequently only discovered post-mortem, so that it may give rise to but few symptoms during life. On the other hand, it is sometimes associated with severe epigastric pain, vomiting and slight hæmatemesis occurring in relation to the ingestion of food. There may be anorexia.

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[1] Cirrhosis ventriculi can only rarely be a primary disease: it is difficult to see how the extensive fibrosis that occurs can result from any ordinary irritant (such as food) taken into the stomach. In some cases diffuse carcinoma has been mistaken for cirrhosis ventriculi. Thus the two specimens shown in Figs. 53 and 54 were catalogued in the Museum as "Chronic Gastritis"; microscopically, however, they are both cancer.

The functions of the stomach are greatly diminished, and the organ cannot be inflated.

The physical signs are the same as those of diffuse cancer of the stomach (see Chapter XVI. p. 482). General dropsy may occur.

Diagnosis.—Inasmuch as the symptoms and physical signs of simple and malignant cirrhosis ventriculi do not materially differ, the diagnosis is mainly directed to discover whether the disease is malignant or not. The only point which is of aid in this direction is the duration of the presence of the oblong epigastric tumour. In simple cirrhosis ventriculi the tumour may be observed during ten or fifteen years (Brinton).

Treatment is that of permanent gastric insufficiency (Chapter. XI.) and of cancer (Chapter. XVI.).

CHAPTER XV.

ULCER OF THE STOMACH, ACUTE AND CHRONIC.

Ulcus Ventriculi Rotundum, Perforans, Corrosivum, Pepticum, Rodens: Ulcus e Digestione: Ulcus Pepticum.

Definition and Varieties.

Ulcer of the stomach is a disease in which, over an area of varying size, there is a destruction of the walls of the organ, tending to involve all the coats of the organ, so that an open sore is left. It exists in two forms: the *acute*, in which the rapid formation of the ulcer gives it the appearance of being punched out of the stomach walls; the *chronic*, in which the edges and base of the ulcer are shelving, so that it is funnel-shaped. Both lead to hæmorrhage and tend towards perforation of the organ, and the extrusion of its contents into the peritoneal cavity. The chronic tends to heal, producing a cicatrix which frequently causes deformity of the organ: the acute may heal, but rarely leads to deformity.

By the definition erosions of the mucous membrane are excluded; they are superficial, and on healing leave a smooth scar which does not lead to deformity of the organ.

Varieties.—The two chief forms are the acute perforating ulcer and the chronic ulcer. The only other disease of the intestinal tract which is related to gastric ulcer both pathologically and clinically is ulcer of the duodenum, which may also be acute or chronic.

ETIOLOGY.

The pathological factors producing ulcer of the stomach are, as will be shown, probably numerous, and although the result as regards the formation of an ulcer may be the same, yet this fact must be borne in mind in considering the etiology of the disease.

Age.—Ulcer of the stomach may clinically be divided into the ulcer of young adults and the ulcer of middle age. Between the ages of fifteen and thirty years, it is most commonly found. In 226 cases of ulcer and cicatrices, collected from post-mortem examinations by Dr. Brinton, the following results as regards the incidence of age were obtained:—

Between 0 and 20 years	20 cases.	Between 50 and 60 years	32 cases.
,, 20 ,, 30 ,,	45 ,,	,, 60 ,, 70 ,,	32 ,,
,, 30 ,, 40 ,,	39 ,,	,, 70 ,, 80 ,,	15 ,,
,, 40 ,, 50 ,,	38 ,,	,, 80 ,, 90 ,,	5 ,,

These figures, however, by no means show the great frequency of cases of gastric ulcer in early adult life. In infancy and childhood, ulcer is rare; it is not till puberty is past that it becomes a frequent disease. In old age, again, it is a rare disease, the tendency to the formation of ulcer appearing to cease soon after middle age. Post-mortem statistics cannot be taken as an accurate means of deciding the age-frequency of ulcer, since it is impossible to tell the exact duration of the disease from an examination of the ulcer. Thus a person may die in middle age of an ulcer contracted during early adult life, or in old age from an ulcer formed in middle age. More accurate statistics are obtained by comparing the age-frequency with the duration of symptoms, although even this method is not strictly accurate, since not a few cases, especially in middle life, are "latent" in regard to the symptoms produced. In 171 cases collected from the records of University College Hospital, in all of which the presence of ulcer was undoubted from the symptoms, the age-incidence was as follows:—

Between 0 and 20 years	15 cases.	Between 40 and 50 years	25 cases.
,, 20 ,, 30 ,,	75 ,,	,, 50 ,, 60 ,,	14 ,,
,, 30 ,, 40 ,,	38 ,,	Over 60 years	4 ,,

These figures give a more correct estimate of the age-incidence of ulcer than those of Brinton, since the duration of the symptoms was known and was taken into account in their compilation.

Sex.—Gastric ulcer is from three to five times as frequent in women as in men. Of the 171 cases occurring at University College Hospital, 144 occurred in women and 27 in men: a proportion of 5·3 to 1. A comparison of the ages at which the disease occurred in the two sexes shows that it is commonest in young women under the age of thirty, and that in men the tendency to ulcer increases towards middle age.

THE AGE-INCIDENCE IN SEX OF ULCER.

	0–20 yrs.	20–30 yrs.	30–40 yrs.	40–50 yrs.	50–60 yrs.	Over 60 yrs.	Total.
Women	14 cases	70 cases	32 cases	15 cases	10 cases	3 cases	144
Men .	1 ,,	5 ,,	6 ,,	10 ,,	4 ,,	1 ,,	27

Its frequency in women cannot be clearly explained, but it is frequently associated with chlorosis (see pp. 400 and 409).

Occupation appears to have but little influence in the production of gastric ulcer. It is said by some to be more common in the working classes than in the well-to-do. This is a difficult point to determine, but the difference in the diet of the two classes may have some influence in its production. The large excess of vegetable food in the diet of the working class may be one of the factors in the causation of the ulcer, although it is not a point on which definite statements can be made.

Relation to other Diseases.—Ulcer of the stomach may exist by itself, and be the sole organic disease present during life or found at death. This is not only so in middle age, from forty to fifty years, but is also the case in young adults. On the other hand, it is frequently associated with other diseases, which may of themselves be fatal or may be actively progressing at the time of death.

In some of the diseases with which ulcer is associated, but little connection can be traced between the stomach condition and the general disease. Thus *ague* and *syphilis* have been supposed to have some relation to gastric ulcer, but the connection between these two conditions and gastric ulcer must be a very remote one. They are both diseases, however, which, if of long standing, produce anæmia and a general lowering of resistance to disease, a condition which predisposes to infection, and may, as will be hereafter discussed, in some cases possibly lead to ulcer.

Tuberculosis is another disease which is more commonly associated with ulcer of the stomach than either ague or syphilis. In a certain number of cases of pulmonary tuberculosis, simple gastric ulcer is found; a tubercular ulcer of the stomach being of great rarity, and only occurring in advanced tuberculosis. The frequency with which gastric ulcer is associated with pulmonary tuberculosis does not warrant the conclusion that this disease predisposes to any great extent to the formation of an ulcer in the stomach. Tuberculosis is not present in cases of gastric ulcer in a greater proportion than the average incidence in individuals generally (Brinton). The conditions in tuberculosis which may be said to predispose to gastric ulcer are the anæmia, which is the result of the disease, and the stomach changes which frequently accompany the disease. How far these stomach changes favour the production of ulcer will be discussed under the heading of pathology (p. 409).

In *portal obstruction* and in *cardiac disease* causing embarrassment to the circulation in the right side of the heart, ulcer of the stomach is sometimes observed. These diseases have been considered as directly concerned in the production of ulcer, since in both there is mechanical congestion of the stomach which may be attended by local accidents (hæmorrhage, thrombosis) directly leading to the formation of an ulcer. How far this is true, it is impossible to say. Ulcer of the stomach is not specially frequent in these diseases, although both portal obstruction and mitral valvular disease have a great effect on the ulcer when this has already been formed, owing to the presence of mechanical congestion of the organ.

With *chronic renal disease*, ulcer may be associated, but there is no evident connection between the two diseases.

With the *chlorosis* of young women, ulcer appears to have more than a fortuitous connection. The larger proportion of cases in young adults occur in women who are the subject of chlorosis and have exhibited one or more exacerbations of the disease. When an ulcer is actually present, the symptoms of chlorosis may be very marked, or there may be a history of an attack of chlorosis one year or more previously, the symptoms of ulcer developing subsequently. Thus the development of a gastric ulcer does not appear to have any immediate connection with the acme of the symptoms of chlorosis. But even in those cases where there is a history of chlorosis with recovery, the anæmia is not completely absent; amenorrhœa, some dyspnœa, and the physical signs of the condition (venous hum and a hæmic murmur) being present. Indeed, whether there are symptoms of chlorosis or not, an examination will usually reveal some signs of anæmia, which may, in some cases, be due to an antecedent hæmatemesis.

Chlorosis and gastric ulcer thus evidently are intimately connected, and their association partly explains the greater frequency of gastric ulcer in women than in men, for chlorosis is a disease of women.

The condition in chlorosis is one of impoverishment of the blood (diminution in the amount of hæmoglobin and of red corpuscles), and also of altered relation between the arteries and the tissues, the vessels being smaller than is requisite for the needs of the tissues (Virchow). (See Pathology, p. 409.)

The condition of *amenorrhœa* has been supposed to have some relation to the formation of gastric ulcer. But little need be said on this point. Amenorrhœa is a symptom either of a local condition (disease in the pelvis), or of some general disease. Patients with gastric ulcer not infrequently suffer from amenorrhœa, but as a rule this is due to the coexisting chlorosis, or it may be a temporary condition due to an antecedent hæmorrhage. Similarly the *puerperal state* has been considered as specially predisposing to gastric ulcer; but as regards the normal puerperal state no facts of any value are forthcoming.

Pyæmia and Septicæmia.—In a certain number of cases of pyæmia and of septicæmia, but chiefly the former, gastric or duodenal ulcers are found. These may be of the chronic variety, which cannot be said to have any direct relation to the acute disease present. But they are more frequently of the acute variety, and multiple; two or three being present. These acute ulcers have a direct relation to the pyæmia or septicæmia, and are in all probability produced by septic embolism.

Burns.—In rare instances burns are associated with acute ulceration of the stomach as well as of the duodenum, and the explanation of their occurrence is probably the same as that in septic conditions. When an ulcer of the stomach is associated with a burn the duodenum is almost invariably ulcerated as well (see Duodenal Ulcer, p. 456).

Brinton attributed great importance to "old age, privation, fatigue, mental anxiety, and intemperance" in the causation of ulcer, but with the exception of old age, the connection between ulcer of the stomach and these conditions must be considered as very distant.

PATHOLOGICAL ANATOMY.

Acute Ulcer of Stomach or Duodenum.—The acute ulcer is usually small, varying in size from half an inch to one and a half inch in diameter, and has in most cases the appearance of being punched out of the walls of the stomach or duodenum. It frequently perforates, the rupture of the peritoneum being round or more usually ragged. The edges of the ulcer are not thickened but are congested, often deeply; and may show petechial spots, which are also not infrequently present in the mucous membrane around. The base of the ulcer when it does not perforate may be smooth or somewhat irregular, and shows in parts small masses of totally necrosed and of necrosing tissue (slough). The appearances of an acute ulcer of the stomach and duodenum are those of an inflammatory process ending in necrosis, such as occurs in acute bacterial infection. Two ulcers are not infrequently found near each other, perhaps somewhat varying in size; and ulcers may be present both in the duodenum and in the stomach. In not very common cases a large chronic ulcer of the stomach may be found, and near it a small acute ulcer which has perforated and been the immediate cause of death.

The situation of acute ulcers is variable. In the stomach they are perhaps most usually situated in the pyloric region, but they are not so frequently situated on the posterior surface as the chronic ulcer. In the duodenum they are situated in the first part, *i.e.* the first horizontal portion, but do not specially choose either the anterior or posterior surface of the gut as their seat, and they may be either transverse or longitudinal.

Chronic Ulcer.—The chronic ulcer of the stomach presents appearances widely differing from those of the acute ulcer. In the acute ulcer the appearances are those of an acute inflammatory and necrosing process; in a chronic ulcer, there is a loss of substance of the walls of the organ in a peculiar manner, with evidences of surrounding chronic inflammation. Some chronic ulcers may have originated as acute, but there is little evidence of this. The anatomy of the ulcer appears to show that it is chronic from the first.

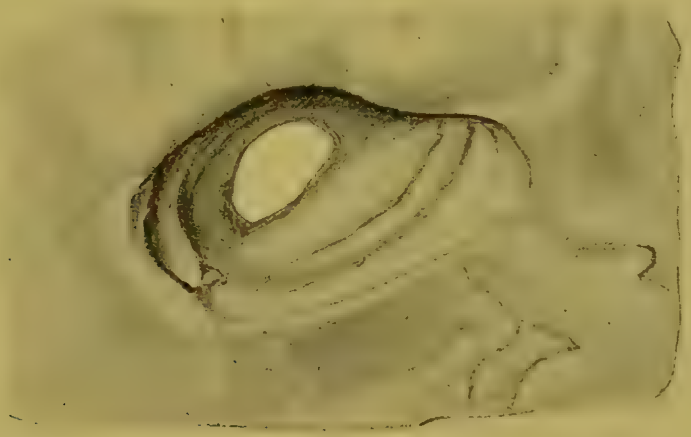

FIG. 35.—Perforated chronic ulcer. (From a preparation in the Museum of the Royal College of Surgeons. By permission of the Council.) Twice the natural size. The ulcer is shaped like an oyster-shell. It is funnel-shaped, and the ridges formed by the submucous and muscular coats are well seen. The peritoneum is perforated by an oval opening with clean cut edges.

Wherever situated the chronic ulcer is funnel-shaped (Fig. 35), the wider end of the funnel being at the mucous membrane, the smaller situated towards the peritoneal coat of the organ. That is, in the open ulcer, the mucous membrane is destroyed to a greater extent than the submucous coat, and this than the muscular coat, while the peritoneum is destroyed over only a small area. The *edges* of the ulcer are often greatly thickened; they may be 1 inch in thickness, and on section this thickening is whitish and translucent in appearance. It is seen to be due chiefly to an increase of the fibrous tissue in the submucous coat, but microscopically the mucous membrane, and to a greater extent the muscular coat and the peritoneum are found thickened and fibroid and form part of the thickened edge of the ulcer (Fig. 36). The *base* of the ulcer is irregular, owing to the ledges of the coats of the organ; it may show here and there small portions of brownish slough, but these are usually absent, and it may be of a uniform brown colour due to the action of the gastric juice on the blood in the superficial capillaries. The base is frequently formed in the centre of the ulcer by the peritoneum alone. This occurs when there is no solid organ (liver, pancreas, spleen) opposite the base of the ulcer. Around this central peritoneal base is seen the muscular coat of the organ, which is recognised by its fine striation. The muscular base of the ulcer is often wide in extent; it appears resistant to the chronic ulcerative process, and the thick circular muscles are more resistant than the thin longitudinal (the oblique not often being recognisable). At the edge of the exposed muscle is the thickened submucous coat with the mucous membrane frequently curved over it, but not undermined. Not infrequently the edge of the ulcerated muscular coat (circular) retracts beneath the thickened submucous and mucous coats, increasing the thickness of the edge, but leaving the ulcer with a smoother base and more vertical edges. An ulcer may also present a shelving and thickened edge round the greater part of its periphery (chronic process), and an abrupt slightly thickened edge round the remainder (more acute process) (Fig. 37). This is seen in old ulcers, and may be taken as evidence of one mode of recrudescence of the ulcer. In other cases the acute process chiefly affects the base of the chronic ulcer, and perforation results. When the ulcer is

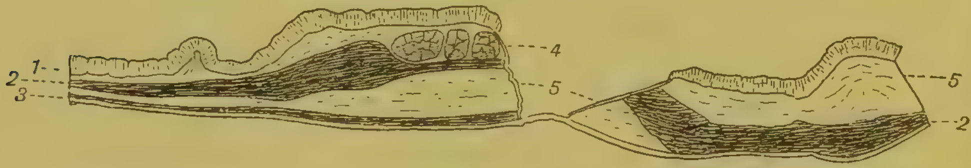

FIG. 36.—Diagrammatic sketch of section of chronic ulcer. 1, mucous membrane; 2, muscular coat; 3, bundle of longitudinal fibres separated from rest of muscular coat; 4, circular fibres hypertrophied, cut transversely; 5, chronic inflammatory thickening.

opposite the pancreas and liver, and in rare cases the spleen, the base of the ulcer is often formed by the surface of these

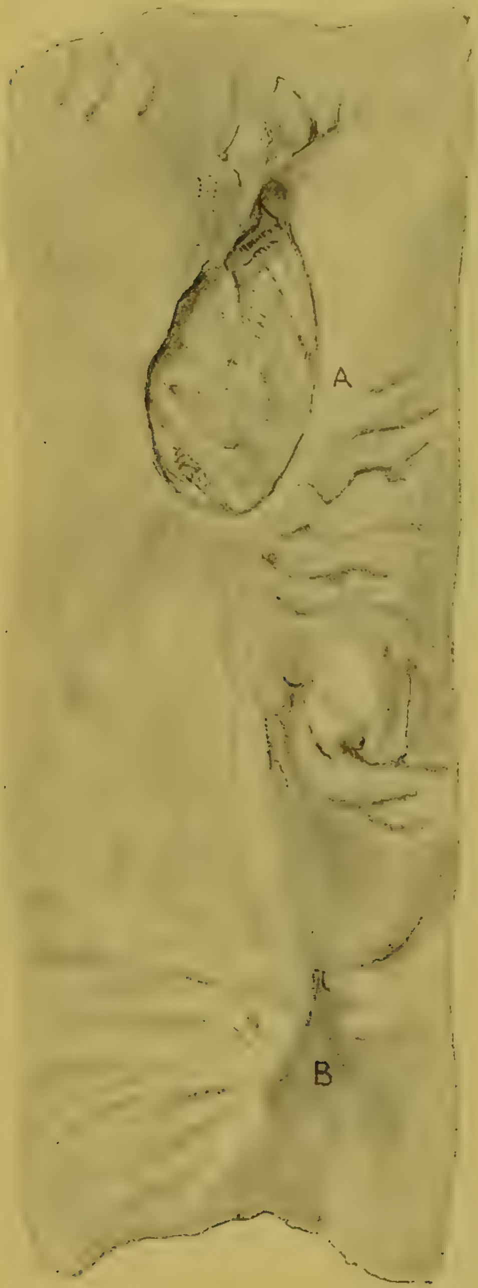

Fig. 37.—Portion of a stomach showing an ulcer at one part (A) and a scar at another (B). (From a preparation in the Museum, University College.) About natural size. The *ulcer* (A) is oval and measures 1⅜ inch by ⅞ inch. It is shallow, and the edge is smooth, but is abrupt at one side (lower part of figure) and shelving at the opposite side. At the latter spot a line across the base of the ulcer represents the edge of the muscular coat. The floor of the ulcer is smooth, but is deeper at the shelving edge than at the opposite side, and here there is a small oval perforation, with shreds of tissue across it. The ulcer crosses the line of attachment of the lesser curvature. The *scar* (B) is situated in the same line as the ulcer, it is roughly triangular and has caused puckering of the mucous membrane over it. From a patient, æt. 38, who died from repeated hæmatemesis.

organs, and cavities (abscesses) may subsequently be formed in these organs.

Adhesions are formed between the ulcer and the solid organ opposite to it, most frequently to the pancreas, less frequently to the liver, and only in occasional instances to the spleen or mesentery (Fig. 38). Owing to the fact that there is no solid organ opposite the anterior surface of the organ, and that the abdominal wall is in constant motion, adhesions of an ulcer on the anterior surface of the stomach to the parietal peritoneum are rare. Adhesions are protective against perforation, and are most effectual when they occur with the pancreas (owing to its immobility), less effectual when they occur with the liver. In the latter case they are liable, when recent, to be ruptured by sudden contraction of the diaphragm (as in coughing and sneezing) or by vomiting.

Shape of the Ulcer.—The chronic ulcer varies in shape to some extent; it is round or oval, or the shape may be that of a horseshoe. The round or oval ulcer (Figs. 37 and 39) calls for no particular description, but the horseshoe-shaped ulcer is peculiar inasmuch as it derives its name from the fact of starting on the posterior surface of the organ, and passing across the lesser curvature and so on to the anterior surface. It is usually a very large ulcer.

Size of the Ulcer.—The size varies from ½ an inch to 1 inch in diameter, or it may be 4, 5, or 6 inches, by 1½ to 2 inches. The largest ulcers are horseshoe-shaped ulcers, and those arising on the posterior surface; they are nearly all found in the pyloric portion of the stomach and about the lesser curvature. The large ulcers are of long-standing. There is no relation between the size of the ulcer and its danger; as even when not more than half an inch in diameter it may cause death by hæmorrhage or perforation.

Site of the Ulcer.—The relative frequency of the locality of ulcer of the stomach is seen by the following statistics given by Dr. Brinton of 216 cases examined post-mortem:—

On the posterior surface	86 cases, or	40 per cent.
On the lesser curvature	56 ,,	26 ,,
At the pylorus	32 ,,	15 ,,
On the anterior and posterier surfaces . .	13 ,,	6 ,,
On the anterior surface only . . .	10 ,,	4·6 ,,
On the greater curvature . . .	5 ,,	2·3 ,,
On the cardiac pouch	4 ,,	2 ,,

By far the greater number therefore of ulcers are found on the posterior surface of the organ and on the smaller curve, and more than three-fourths of the ulcers are found in the pyloric portion of the stomach.

Results of the Ulcer.—An ulcer may either (1) cicatrise or (2) perforate. Perforation may be either a part of the chronic process, or may be an acute process added to the chronic, or final perforation may be due to a rupture of the thin peritoneal coat of the ulcer (Fig. 37).

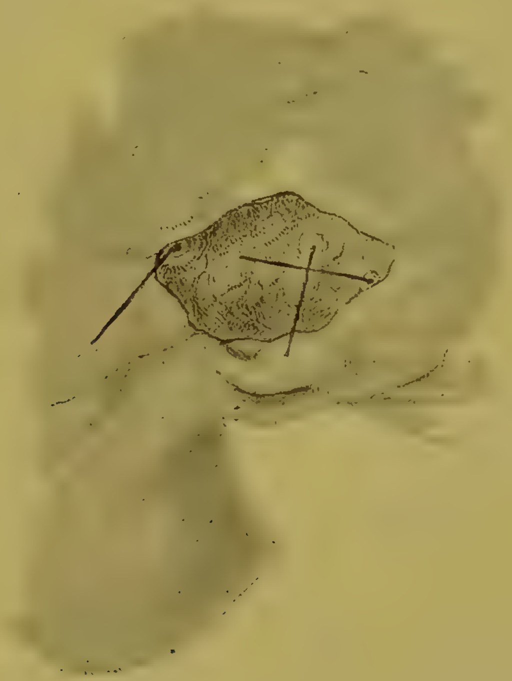

Fig. 38.—The pyloric end of a stomach, showing an ulcer on the posterior wall. (From a preparation in the Museum, University College.) About natural size. The ulcer measures $\frac{3}{4}$ by $\frac{9}{16}$ inch. It is oval, with deeply-cut overhanging margins: complete perforation of the coats of the organ having taken place, so that there is a small cavity outside the organ. The floor is formed by thickened omental or mesenteric tissue. Bristles are placed in three openings, which are erosions into large arteries, branches probably of the coronary artery. From a young male who died from repeated hæmatemesis.

Cicatrisation of the ulcer is of very frequent occurrence; in Brinton's statistics, to 156 open ulcers there were 147 scars, a proportion of 13 to 12. According to the size of the ulcer there is subsequent contraction of the scar, so that there is frequent deformity in the organ produced. The scar left by an ulcer may be a smooth area surrounded by a puckering of the mucous membrane (Fig. 37), or there may be simply a puckering of the mucous membrane as in the healing of small ulcers (Fig. 40).

According to the site of the ulcer there are peculiarities as

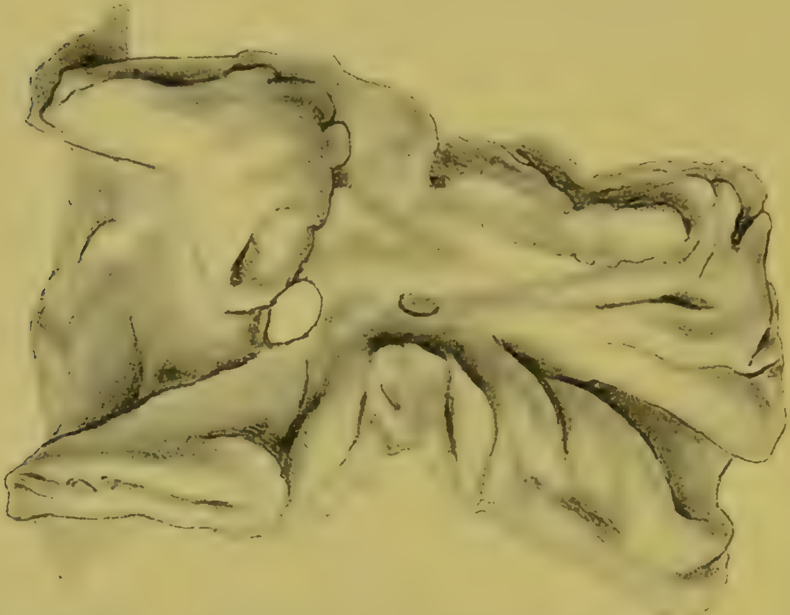

FIG. 39.—Perforated ulcer at the pylorus. (From a preparation in the Museum of the Royal College of Surgeons. By permission of the Council.) About natural size. The duodenum is to the right, and just within the pylorus is seen an oval perforation of the stomach wall. This perforation constitutes the whole of the ulcer. The pylorus is constricted, and there is puckering of the duodenum.

regards its results, whether as leading to perforation or as

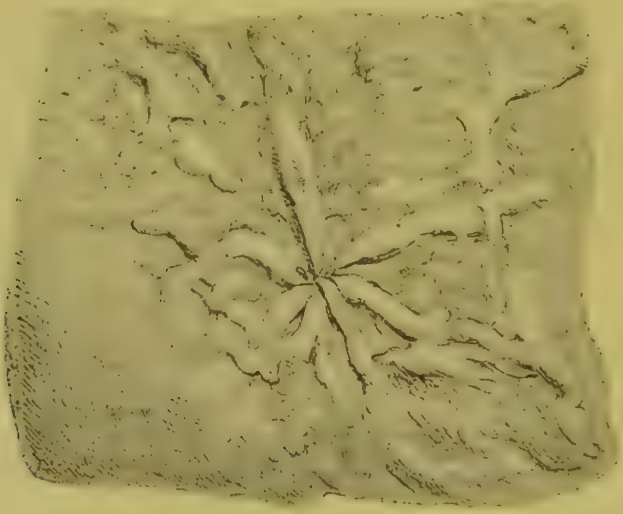

FIG. 40.—Portion of the stomach of a boy, showing the scar of an ulcer following an extensive burn. There is a star-shaped puckering radiating from the ulcer which had quite healed. The peritoneum was normal. (From a preparation in the Museum, University College.)

producing deformity of the organ. Ulcers on the posterior surface have as their base the pancreas, and may lead to subphrenic abscess. Ulcers along the smaller curve lead to contraction of the organ, or rather approximation of the cardiac and pyloric orifices; they may also lead to local dilatation of the pyloric pouch. Ulcers of the pylorus lead to stricture of the orifice with subsequent hypertrophy and dilatation of the organ. The horseshoe-shaped ulcer may lead to hour-glass contraction of the stomach; and those ulcers situated on the anterior surface or in the cardiac pouch lead to perforation of the organ with general peritonitis or subphrenic abscess, and those near the cardia may lead to stricture of the orifice (for further details, see Complications of Ulcer, p. 440).

PATHOLOGY.

The pathology of ulcer of the duodenum and stomach is very obscure, and for an explanation of the peculiar construction of the chronic ulcer, the special structure and functions of the stomach and first part of the duodenum must be considered, since this kind of ulcer exists nowhere else in the alimentary tract.

In discussing the etiology of gastric ulcer (see also Duodenal Ulcer, p. 454), it was seen that there was only one condition of the body which might be said to bear a close relation to the formation of ulcers in the duodenum and stomach; this was the existence of pyæmia or septicæmia, or of an allied condition in extensive burns of the skin. In the majority of ulcers, both of the stomach and duodenum, pyæmia and septicæmia are, however, not primary causes, and there is no etiological association which is explanatory of their formation. The age of the patient, as has been seen, does not aid, because ulcers occur in the young and middle-aged. Although sex is evidently of some importance since gastric ulcers are twice as frequent in females as in males, yet the differences between the male and female organism does not greatly help, especially as in duodenal ulcer, the sex-relation to the disease is reversed. The female goes through physiological and pathological crises from which the male is exempt: in young womanhood, the development of chlorosis; in married life, the occurrences of pregnancy, parturition, and lactation; in middle life, the occurrence of the climacteric, after which the physiological processes of male and female are practically identical. These differences between the female and male bear no distinct relation to the disease, and it is probable that for an explanation of the formation of the gastric and duodenal ulcer, mainly a local cause in the stomach or duodenum must be looked for.

Causes of Ulceration generally.—The pathology of ulceration does not differ from certain other processes which occur in solid tissues. The chief event occurring is the death of the tissue. In an ulcer this dead tissue is cast off, leaving an open sore, whereas in a solid organ it is retained in the cavity or absorbed. Necrosis of the tissue therefore is the primary factor in the production of an ulcer. Usually, however, added to this there is an irritant which may act for a longer or a shorter time before the ulcer is produced.

There are three common causes of the death of the tissue which precedes ulceration.

1. *Mechanical and Chemical Causes.*—Corrosive poisons by directly destroying the tissue lead to ulceration; and an injury to the mucous membrane, which is subsequently exposed to the continued action of an irritant, will also lead to an ulcer.

2. *Interference with the Vitality of the Tissue.*—The vitality of a particular part of the mucous membrane may be diminished by local and chronic disease or by interference with the circulation over a certain area. This latter usually occurs by the means of thrombosis or embolism. Thrombosis takes place in connection with disease of the vessels and in association with a diminished quality of the blood and a slowing of the local circulation; embolism may be infective or non-infective, and is usually capillary.

3. *Bacterial Infection.*—In acute infective processes of the mucous membrane, where there are the signs of active inflammation, ulceration may result, as in typhoid fever, dysentery, etc. A similar result occurs in chronic infective processes as in tuberculous ulceration of the intestine. But there is another kind of bacterial infection which is unassoci-

ated with the signs of active inflammation, and which is best called bacterial necrosis. It has not been fully studied, but it undoubtedly possesses great pathological significance, and may serve as an explanation of some of the pathological processes which occur in the gastro-intestinal tract. Bacterial necrosis has been more commonly found in animals than in man, although it does exist in human beings. In cattle, pigs, guinea-pigs, especially the two latter classes of animals, bacterial necrosis is extremely common in the mucous membrane of the alimentary tract and in the lympathic glands connected with it, as well as in the liver and spleen. The process is characterised by the invasion of bacteria usually into the lower depths of the mucous membrane, by their growth, and by the subsequent necrosis of the tissue. Surrounding the area of necrosis not uncommonly is an infiltration of leucocytes which may organise into fibrous tissue.[1] An

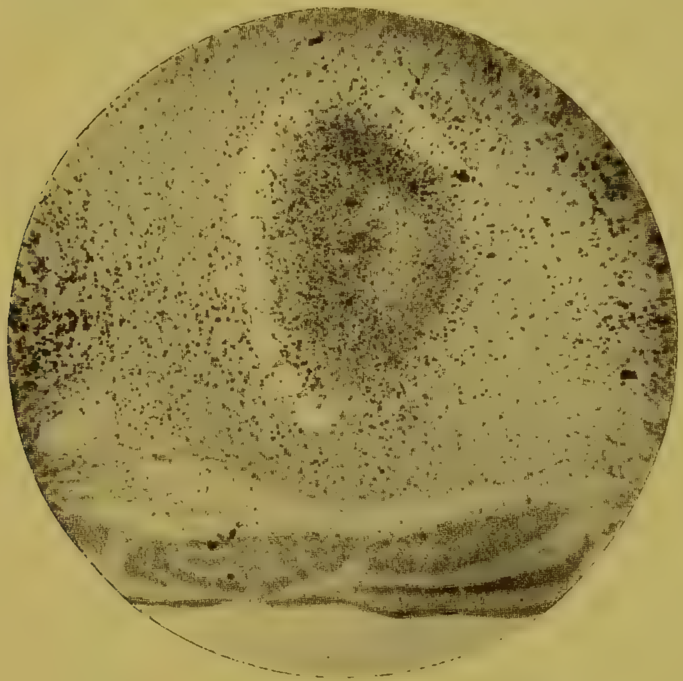

FIG. 41.—Bacterial necrosis in a Peyer's patch of the intestine of the guinea-pig. From a photograph, × 60. The lower part of the patch is seen with the muscular and peritoneal coats attached. In the patch is a discrete area separated from the normal surrounding lymphoid tissue and composed of cells undergoing necrosis. The dark masses in the necrosed area are groups of bacilli. There were no tubercle bacilli.

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[1] "Report of the Royal Commission on Tuberculosis," part ii., *Enquiry* ii.

example of this is shown in the accompanying figure (Fig. 41), which represents a section of a Peyer's patch of the intestine of the guinea-pig. In the lower depths of the Peyer's patch may be seen a necrotic area with dark masses in the centre; these are distinguished as groups of bacilli when seen under a higher power than is here represented. This is an example of the first changes in bacterial necrosis. In the extension of the process, the areas of necrosis increase, until a comparatively large part of the mucous membrane is affected. Ulceration may result. The changes in bacterial necrosis in the lymph glands, liver, and spleen are similar to those shown. Bacterial necrosis may be caused by different kinds of cocci, both strepto- and staphylo-cocci, and by various forms of bacilli, but the differentiation of these micro-organisms has not as yet been sufficiently studied for a classification of them to be made.

Speaking generally, therefore, an ulceration of a mucous membrane may be either infective or non-infective, and it is with such classification as this in mind that ulceration of the stomach and duodenum will be discussed.

Causes of Ulceration in the Stomach and Duodenum.—In the localities in which ulceration of the stomach and duodenum is most commonly found, that is in the stomach over the pyloric area and in the first part of the duodenum, no hydrochloric acid is secreted by the glands of the mucous membrane. Hydrochloric acid is present in the stomach contents during digestion, and bathes these parts, but it is an important fact in the pathology of ulceration that the glands of the pylorus do not secrete hydrochloric acid. The actual occurrence of an ulcer, *i.e.* loss of tissue resulting in the production of an open wound after the death of the tissue, has been ascribed to the digesting action of the gastric juice; and this has been expressed in the names sometimes given to the ulcer—*ulcus pepticum* and *ulcus e digestione.* That the gastric juice has some action in the production of the ulcer peculiar to the stomach and first part of the duodenum is probable from the fact that these ulcers present a great anatomical contrast to those occurring in the rest of the small intestine. These latter are almost invariably directly infective, as, for example, typhoid and tubercular ulceration and the ulceration in "tropical" diarrhœa, or they are due to a carcinomatous new growth. But the gastric juice does not itself produce an ulcer, since it has no action on the healthy living membrane, and a mechanical injury of the mucous membrane of the stomach readily heals, as has been shown by experiment. There must be some antecedent change in the mucous membrane whereby the vitality of the tissue is damaged. In such a case the gastric juice will readily remove the dead tissue by its digestive action, and so produce the ulcer of the stomach. Once the ulcer is formed, the gastric juice and the process of digestion of the mixed food prevents its healing, and leads to its chronicity. Before, however, the formation either of a chronic ulcer or of an acute ulcer there must be death of the tissue, and it is as to the causes of this primary necrosis that knowledge is very imperfect, and about which opinions have so much differed.

The following different causes of the necrosis of the tissue preceding ulceration of the stomach and duodenum may be considered:—

1. *Injury.*—Poisoning with corrosives, mineral acids, and caustic alkalies may lead to the formation of a chronic ulcer, resembling anatomically that produced by other causes. In these cases the acid or alkali destroys part of the mucous membrane to a greater or less depth, and the ulcer then enlarges partly by progressive death of the tissue and partly by the action of the gastric juice. Such ulcers may occur at the cardiac orifice, but more usually at the pylorus.

2. *Pressure Necrosis.*—Ulcers may be due to pressure, a so-called pressure necrosis of the mucous membrane resulting. It is sometimes found that an aneurysm which is adherent to the cardiac pouch has produced an ulcer of the mucous membrane corresponding to the area of adhesion of the tumour. In such a case as this the necrosis has been secondary to the obliteration of the vessels in the outer coats of the organ, associated with an interstitial fibrosis.

Another form of pressure necrosis I have seen occurring in the mucous membrane itself in the case of submucous fibro-myomata of the stomach. These small tumours, ranging in size from a half to one centimeter in length and about half that width, are not unfrequently present in the submucous tissue in the stomachs of people who have died in middle age. As a rule their presence is of but little significance, but in one case a small ulcer on the surface of one of

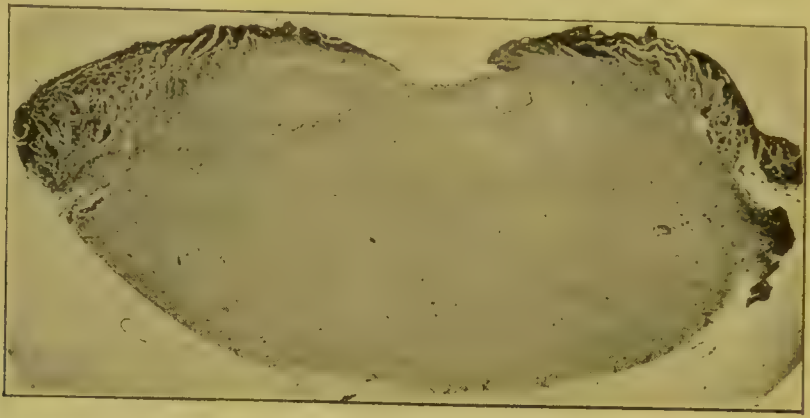

FIG. 42.—Fibro-myoma of the stomach, with ulceration of the mucous membrane. From a photograph, × 9. The specimen was taken from a male of middle age, who died from perforation of a chronic gastric ulcer of long-standing. The fibro-myoma forms an oval tumour in the submucous coat with the mucous membrane adherent to it and undergoing necrosis. In the centre of the tumour, the mucous membrane has disappeared, leaving a shallow ulcer, the base of which is formed by the tumour. See also Figs. 43 and 44. The dotted line on the right represents the part from which Fig. 43 was taken. From a preparation hardened in Marchi's fluid and stained with logwood.

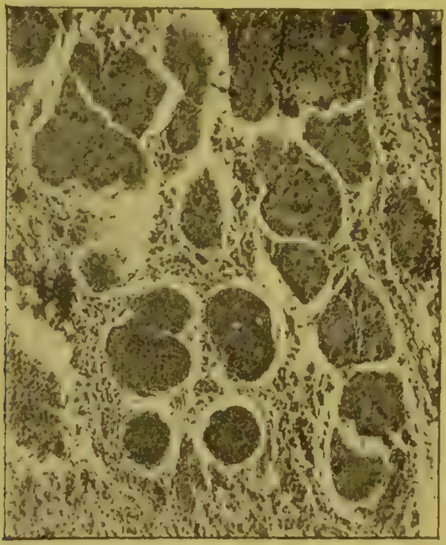

FIG. 43.—Mucous membrane of the specimen represented in Fig. 42. From a photograph, × 90 The gastric glands are separated by fibroid tissue containing a large number of small cells. The glands themselves are in a state of atrophy: the outline of the cells is lost, the nuclei are indistinct, and the body of the cells is becoming granular and vacuolated. From a preparation hardened in Marchi's fluid and stained with logwood.

these tumours was seen, as represented in the following figures (Figs. 42, 43, 44).

In the first figure it will be observed that the oval tumour is adherent to the mucous membrane above, and that the mucous membrane is completely absent over the centre of the tumour. An open ulcer, two millimeters in diameter, was to be seen in that situation in the recent specimen. On each side of the ulcer the mucous membrane shelves off and is in a state of more or less complete necrosis, showing that eventually the ulcer would have been much larger; and it is probable that the whole of the small tumour would have sloughed away leaving a deep but small gastric ulcer.

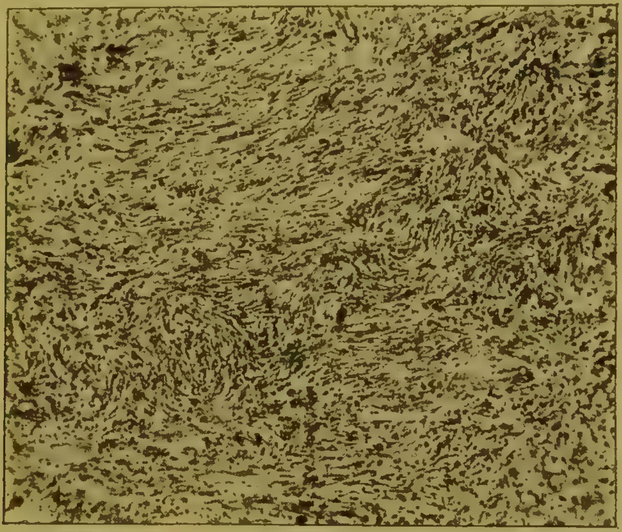

Fig. 44.—Fibro-myoma of the stomach (Fig. 42). From a photograph, × 90. The figure shows the interlacing bundles of unstriped muscle fibre, with the characteristic elongated nuclei. Some of the fibres are cut transversely. From a preparation hardened in Marchi's fluid and stained with logwood.

The second figure shows the mucous membrane under a higher power and in a state of interstitial fibrosis; the glands are compressed and have lost their typical structure, the cells being fused together and the nuclei irregular and in a state of atrophy.

The third figure shows the structure of the tumour itself as being a fibro-myoma, the nuclei of the spindle-shaped muscle fibre being well seen.

This mode of formation of gastric ulcer is not common, but that it does occur must be recognised by the results of the examination of the above case which occurred in a patient who had a large chronic ulcer situated near the pylorus. The causes of the necrosis of the mucous membrane in the above case were no doubt due to adhesion of the tumour to the mucous membrane and subsequent interstitial fibrosis of the membrane, and finally death of the part. An example of fibro-myoma from another case in which this adhesion and subsequent necrosis did not occur is shown in Fig. 45, where

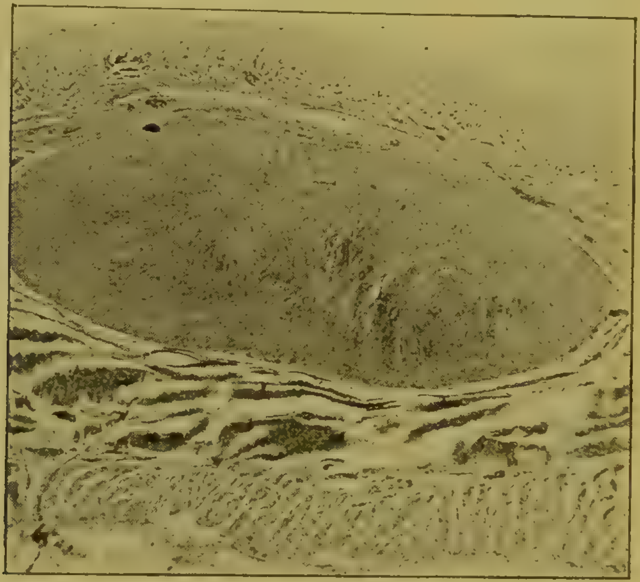

FIG. 45.—Submucous fibro-myoma of the stomach, which has not led to ulceration. From a photograph, × 16. The specimen was taken from a male of middle age, who died from the rupture of an aortic aneurysm. The stomach was otherwise quite normal. The oval tumour, which has the same structure as that represented in Fig. 44, lies in the submucous coat, being separated from the mucous and from the muscular coat by loose connective tissue. The tumour was freely movable beneath the mucous membrane. From a preparation hardened in Marchi's fluid and stained with logwood.

a small tumour is seen to lie encapsuled in the submucous tissue, the mucous membrane being intact over it.

3. *Thrombosis and Embolism.*—Another cause of death of the tissue must be ascribed to changes in the vascular system by which the vascular supply over a greater or less extent is more or less completely cut off. This theory is the one promulgated by Virchow for the explanation of gastric ulcer, and it has been accepted more or less generally. Virchow pointed out that the shape of the chronic ulcer of the stomach was practically

the same as that of the infarcation of the lung or kidney, where the condition of hæmorrhage and subsequent necrosis is due to a blocking of the end artery by means of a clot, and that if there were thrombosis in the larger arteries running between the muscular coat, the necrosis of the mucous membrane following would take the shape assumed by the chronic ulcer of the stomach. The blocking of the vascular supply over a certain area of the stomach may be due to thrombosis or to embolism, and the emboli may be of two kinds, either infective (septic) or non-infective.

In adapting these ideas to the explanation of ulceration of the stomach and duodenum, very great difficulties are experienced. With regard to the acute ulcer, the difficulty is not so great as with the chronic variety. Taking the case of the formation of a duodenal ulcer when there are extensive burns of the skin, it is seen that the duodenal ulcer is formed during the second period of the burn, that is the period of inflammation when the skin sloughs are separating and consequently there is a great danger of emboli being carried to various parts of the body, and it does not appear improbable to ascribe the occurrence of duodenal ulcers in burns to capillary embolism of the mucous membrane.[1] This conclusion is rendered more probable from the fact that acute duodenal ulcer and acute gastric ulcer both occur in the course of a pyæmia or a septicæmia, more usually the former, in which it is known that numerous emboli are frequently sent into the circulation. It does not appear likely that the acute ulcer of the stomach or duodenum can be ascribed to any other condition than that of an acute bacterial infection.

With regard to the chronic ulcer it is probable that in some cases it arises as an acute ulcer, but in the majority of instances it is chronic from the first.

The occurrence of thrombosis as an explanation of the death of the tissue preceding ulceration is in this case impossible of demonstration and very difficult of explanation.

It must be borne in mind that gastric ulcer is most usually found in conditions of the body in which thrombosis is uncommon, there being no acute infective disease or convalescence from such disease to explain the occurrence of thrombosis or embolism. In young women, for example, the subject of chlorosis, chronic gastric ulcer is of great frequency, but in chlorosis there is no special tendency to thrombosis, and there is no reason why such patients should not show thrombosis as well in the femoral vein or in the veins of the intestine as in the stomach. The occurrence of thrombosis therefore is improbable as an explanation for the majority of cases of chronic gastric ulcer that occur in young adults and especially in women. It cannot of course be denied that such an accident as thrombosis in an artery of the stomach might occur in the course of chlorosis; but it must be a rare occurrence, and there is no special condition of the stomach, either as regards the arteries or disease of the walls which would predispose to thrombosis in the case of chlorosis. Again it has been supposed that in some cases of gastric ulcer the initial thrombosis has occurred in varicose veins. Varicose veins of the stomach are, however, rare, and do not as a rule exist in young adults. Again atheromatous degeneration of the arteries as leading to thrombosis has been instanced as a cause of chronic ulcer.[1] Here, again, there is no explanation of cases that occur in young adults, and especially in young women. Atheromatous disease of the vessels, although it does occur in the stomach, is seen, like atheroma of the other vessels of the body, towards middle age. It may perhaps be a factor in the causation of the ulcer of middle age, but it cannot be one in producing the ulcer of young adult life.

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[1] Erichsen's "Surgery," edited by Marcus Beck, vol. i. p. 390, 9th ed., 1888.

4. *Bacterial Necrosis.*—Without denying, therefore, that simple thrombosis may in some cases lead to ulcer, it appears to me as highly probable that many cases of chronic ulcer of the stomach may originate as a bacterial necrosis. And, indeed, cases of chronic ulcer occur in individuals who are in a condition of non-resistance to the invasion of the tissues by bacteria, and the localities in which chronic ulcer of the stomach most

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[1] In some records the fibroid thickening of the arteries in the neighbourhood of the chronic ulcer has been mistaken for a primary disease of the vessel. It is not primary, but secondary to the chronic inflammation: it is a result, not a cause, of the ulcer.

usually occurs, that is the pyloric area, is that in which the antiseptic hydrochloric acid is not secreted by the glands. This last fact is of great pathological significance; the hydrochloric acid of the gastric juice is a powerful antiseptic insomuch that in cases of normal digestion it prevents the development of bacteria in the stomach, although they are swallowed in great numbers with the food. These bacteria are not in all cases killed by the gastric juice, as they are found in large quantities in the lower part of the small intestine and in the large gut. In conditions where the stomach still secretes hydrochloric acid, if a bacterial infection occurred, one would suppose that it would take place, not at the cardiac area where the glands are constantly secreting the acid, but in the pyloric area where the glands do not secrete the acid, but are only bathed on the surface by the acid gastric contents. So that if the bacteria entered the mouth of the glands, the channel by which infection would occur, they would not meet with any antiseptic acid to prevent their development and their invasion of the tissue. Similar remarks apply to the conditions in the first part of the duodenum.

As regards the occurrence of ulcers of the cardiac area the invasion of bacteria might occur under two conditions—in the state of rest of the glands, *i.e.* in the intervals of digestion, and when there is a deficiency or an absence of secretion of the hydrochloric acid. It, therefore, seems probable that in not a few cases of gastric ulcer the primary necrosis may be due to the invasion of bacteria, the subsequent ulceration and extension of ulceration being aided by the action of the gastric juice.

In the subacute or chronic ulcer of the duodenum, occurring in guinea-pigs, I have several times examined the separating slough, and it has invariably shown the presence of a large number of bacteria in it, but whether as the cause or the result of the sloughing it is impossible to say. The duodenum of the guinea-pig commences by a pouch which would favour the invasion of bacteria. The ulcer is anatomically exactly like that occurring in man, and may cause death by perforation and general peritonitis.

Alterations of the Functions of the Stomach in cases of Ulcer.

—These have already been considered (Chapter IV.), but they may again be summarised—

Hyperacidity.—In a large number of cases of ulcer, usually in the early progressive stage, there is hypersecretion of hydrochloric acid in the gastric juice, and this hyperacidity which has been well established by the researches of Riegel and Jaworski and Korczynski is no doubt due partly to the actual presence of an ulcer which acts as an irritant and stimulant to the secretion of hydrochloric acid, and partly due to the presence of gastric irritation induced by the food. Hyperacidity due to organic acids, the product of bacterial fermentation, is of rare occurrence in gastric ulcer.

Diminished Acidity.—In some cases there is a diminished secretion of hydrochloric acid due either to the presence of gastric insufficiency as in cases of pulmonary tuberculosis, or to the existence of chronic catarrh, which, however, is a rare event in gastric ulcer. In the later stages of prolonged ulcer there may be a great diminution of the amount of hydrochloric acid secreted, especially when there is permanent dilatation of the organ.

Pepsin.—The secretion of pepsin is not much altered in the early stages, and may be actually increased. In the later stages it diminishes like the hydrochloric acid.

In not a few cases of ulcer the secretion of gastric juice is within the normal limit.

Movements.—The movements of the stomach are more frequently affected than the secretion, and this is especially so in cases associated with chlorosis and tuberculosis where the stomach may be actually in a state of atony. The result of this is delay of food in the stomach. In other cases there are paroxysmal contractions of the stomach due to muscular irritability set up by the condition of the ulcer and of the nervous system.

Absorption does not appear to be much affected in gastric ulcer unless there is coexisting catarrh or dilatation of the organ.

To sum up, therefore, the chemical processes of the digestion are either normal or actually more energetic than normal, one of the chief defects being deficiency in the movements of the organ.

Added to the changes above mentioned there may be a condition of gastric irritation or gastric insufficiency, and dilatation may be present with the signs of bacterial fermentation. When there is no dilatation, however, bacterial fermentation of the food is a rare event in gastric ulcer. The vomited matters usually consist of the undigested or partly digested meal containing hydrochloric acid, sometimes in abnormal percentage; containing pepsin and the curdling ferment, but containing no mucus and no sarcina or bacteria. The vomited matters, as will be seen, differ greatly from those present in carcinoma ventriculi.

SYMPTOMS.

Ulcer of the stomach in its typical form is shown by a combination of symptoms which is characteristic of the disease, viz. localised pain after food, vomiting, hæmatemesis and melæna. In addition to these symptoms there may be those of gastric irritation and great irritability of the stomach. Some cases of ulcer do not show this typical combination of symptoms, and may be called irregular or latent, so that the symptoms of the disease can be classified into two groups as to whether they are *typical* or *latent.*

In addition to the symptoms referable to the stomach condition there are the signs of the general disease with which ulcer may be associated. In *chlorosis,* for example, there are signs of anæmia, such as pallor of the face and mucous membranes, amenorrhœa, and a deficiency in the red corpuscles and in the amount of the hæmoglobin they contain, as well as the physical signs of the condition, namely a hæmic murmur and venous hum. Such symptoms and signs are aggravated by the occurrence of a recent hæmatemesis. In *cardiac disease* there are the signs of obstruction or incompetence of the valves; in *renal disease* there is albuminuria, high arterial tension, hypertrophy of the left ventricle, and retinal changes. In *tuberculosis,* usually of the lungs, there are the symptoms and signs peculiar to the condition. These diseases need not be further particularised, but their association with gastric ulcer must always be remembered and their existence looked for.

In not a few cases, especially in the ulcer of middle life, there is no general disease present.

Typical Symptoms.—The general symptoms due to the condition of ulcer of the stomach are extremely few. The general appearance is fairly good, the patient being well nourished with a moderate amount of subcutaneous fat. There is no fever, and the presence of fever may indicate either perforation and its results, or pulmonary tuberculosis or other febrile disease, but it is not due directly to the ulcer in the stomach. Not a few patients show an expression of pain in the face, which is not, as has been supposed, diagnostic of ulcer, because it does not differ from that in other cases where pain is due to visceral disease and is chronic in its course.

In long-standing ulcer the patient shows the signs of the prolonged disease and tends, as it is said, to develop a "cachexia." The patient is thin and weak, with a pale face, and anxious, tired, and painful expression of countenance. The muscles are irritable on tapping (myotatic irritability), and the blood shows a great reduction in the number of red corpuscles and the amount of hæmoglobin they contain. This condition, which varies in degree, is brought about by the long-continued pain, by the starvation induced by the fear of eating and by repeated vomiting, and by the loss of blood in repeated hæmatemesis or melæna. In some cases it is associated with permanent dilatation of the organ.

Symptoms Referable to the Digestive System.—Pain is one of the most characteristic symptoms of ulcer of the stomach. Its causation is various, but its primary cause is the presence of an open sore in the stomach wall. It is aggravated and often first brought out by the ingestion of food, especially when solid and indigestible, and of irritating food articles such as spices, alcoholic drinks, tea, and hot drinks of all kinds. During the digestion of a meal the pain is aggravated by the movements of the organ, and by the hydrochloric acid secreted, whether in normal or abnormal amount; and it lasts as a rule in that aggravated form till the stomach is empty. Sometimes continuous pain is produced by distension of the organ, in which case, no doubt, it is the stretching of the ulcer which aggravates the pain. It is relieved by vomiting or by emptying the stomach, or by the expulsion of the stomach contents through the pylorus. It is sometimes alleviated by posture, and the relief so obtained is supposed to result from the contents of the organ being removed from the surface of the ulcer. Thus, in an ulcer near the pylorus relief may be obtained by lying on the left side, the stomach contents going into the cardiac pouch; relief may be obtained in the prone position in ulcer of the posterior surface; and in the supine position in ulcer of the anterior surface. Relief by posture, however, is not of very frequent occurrence, with the exception of that general relief which the recumbent position gives in most cases of pain.

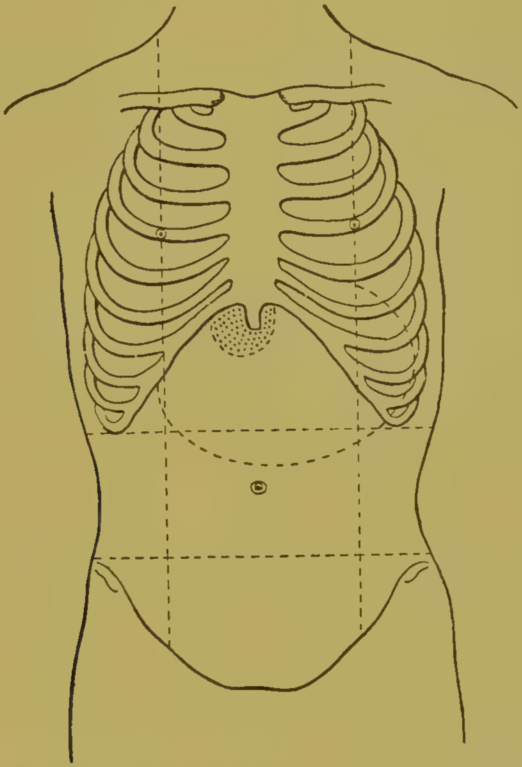

FIG. 46.—Area of epigastric tenderness in gastric ulcer. The outline of the dilated stomach is shown by the dotted line. Female, aged twenty-two years; duration of illness, four months.

In the great majority of cases the pain is increased by pressure, but in some few instances pressure on the epigastrium or doubling up of the body relieves the pain.

Seat of the Pain.—There are two positions in which the pain is felt in gastric ulcer; in the epigastric region and in the back. In either case the pain is localised, and there is local tenderness. The localisation is sometimes very circumscribed, and it may be observed over an area of only 1 inch in diameter; in other cases the area is larger—even 2 inches in diameter. The pain may be more diffuse after a recent hæmatemesis. In the back the pain is usually localised to a region on the left of the spine opposite the tenth to the twelfth dorsal and first lumbar vertebræ. Brinton gives as the upper limit of the dorsal pain the eighth dorsal vertebra, and this is true in some cases.

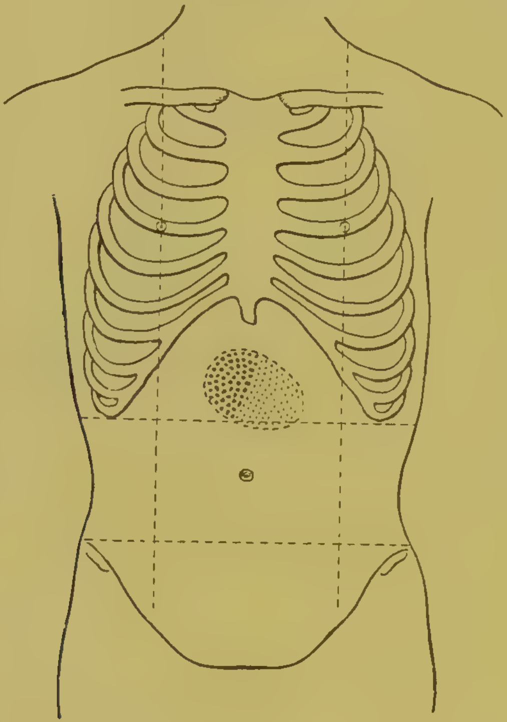

Fig. 47.—Area of epigastric tenderness in gastric ulcer. Over the part darkly shaded, the tenderness was well-marked. Female, aged twenty-seven years; duration of illness, two months; recent hæmatemesis.

The seat of the pain is marked by a local tenderness which may occupy the following positions. Usually it is observed over a small area above and to the right of the umbilicus (Fig. 47). In other cases the tender area is between the umbilicus and the xiphisternal notch. It may be situated at the notch as in the diagram (Fig. 46), which was taken from a case in a female, aged twenty-two, the subject of chlorosis, who gave a history of hæmatemesis four months previously, and a present history of localised pain after eating, in the position of the area of tenderness. There may be tenderness in the left hypochondrium, and one or more areas of tenderness over the stomach region, although this is not common. It is more frequent to observe an area of greater tenderness situated in a slightly tender, larger area as in the diagram (Fig. 47) from a female patient, aged twenty-seven, the subject of anæmia due to hæmatemesis eight days previously; there was also a two months' history of epigastric pain after eating. The epigastric pain and local tenderness are not commonly absent in cases of gastric ulcer, and even in those cases where pain is not complained of, local tenderness may be elicited by deep palpation.

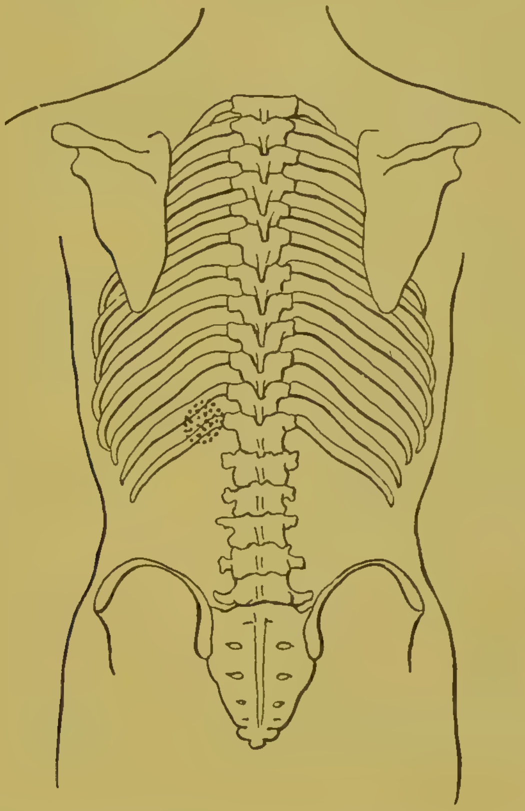

FIG. 48.—Diagram showing the area of dorsal pain and tenderness in gastric ulcer. The area is sometimes larger than is here represented (see text).

The pain in the back is, like the epigastric pain, localised and is shown by local tenderness. It is frequently present, and may be observed when localised epigastric tenderness is doubtful. It is sometimes very circumscribed, viz. on the left side of the twelfth dorsal and first lumbar vertebræ. It is sometimes more diffuse; over an area of 3 or 4 inches in diameter, on the left of the lower dorsal spine, and it may even be present to the right of the spine. Pressure on the epigastrium in some cases produces dorsal pain and tenderness, and both these are aggravated or produced by the presence of food in the stomach.

Local tenderness in gastric ulcer has certain characteristics which are of great value in the recognition of the disease. It is sometimes elicited by superficial palpation of the epigastrium, but in many cases, moderately deep palpation must be resorted to. Deep palpation, as in pressing the hand nearly to the region of the spine, is never to be resorted to, not only because of the danger of inducing hæmorrhage or perforation, but because it fails to bring out the characteristic tender area in gastric ulcer, and tenderness may be elicited owing to the presence of disease in the neighbouring organs of the abdomen or thorax. The characteristics of the local tenderness in gastric ulcer as elicited with care in the manner described are:—

1. The area of tenderness is the region where the patient experiences the greatest pain after eating or drinking.

2. The area of epigastric tenderness persists in its circumscribed form without shifting its locality during the whole course of the illness; as long indeed as the patient has any symptoms, and in not a few cases, after the chief symptoms have disappeared (*e.g.* the pain after food) during the progress of treatment. In recrudescence or relapse of the ulcer, the same area of tenderness is again discoverable, although it may have disappeared previous to the relapse.

Character of the Pain.—The pain varies somewhat in character. In the early stage of ulcer it may be simply epigastric distress, associated with localised tenderness, but it is usually of a heavy, boring character and not sharp or shooting except in rare cases. In the back the pain is of the same character, and the epigastric pain often, as patients express it, "goes through to the back."

Onset of the Pain.—Actual pain is not usually felt by patients with gastric ulcer when the stomach is empty. When there is continuous pain, it is usually due either to the stomach not being empty as in great delay of the food, to distention by gas, or to a condition of nervous irritability kept up by injudicious feeding or by a general condition of the body such as chlorosis.

The pain comes on at two different periods after a meal; either immediately (*i.e.* in a few minutes) or one or two hours later. In cases where the pain comes on directly after the meal, which is the most common condition, the causation must be ascribed to the solid food irritating the ulcer directly, as well as to the movements of the organ which begin immediately food is taken; the pain is then kept up partly by the movements and partly by the secretion of hydrochloric acid until the digestion is completed or vomiting occurs. In cases where the pain comes on some time after food the chief conditions producing the pain are the increasing movements of the organ, and the gradually increasing acidity of the stomach contents. Some of the cases in which there is delay in the onset of pain are no doubt those in which the ulcer is situated just near the pyloric orifice, and the pain occurs when the acid chyme is expelled into the duodenum, as well as cases of duodenal ulcer, in which the acid chyme irritates the surface of the ulcer when it enters the duodenum.

Duration of the Pain.—The pain lasts for a varying time. In some cases vomiting occurs almost immediately, giving relief; but when there is no vomiting the pain may last from four to six hours, causing great distress, until indeed the stomach contents are expelled through the pylorus.

Variations in the Pain.—In some cases the pain is paroxysmal, and it may be aggravated at the menstrual periods or by the presence of chlorosis or tuberculosis. In other cases there is no epigastric or dorsal pain, and there is the pain in the chest after eating solid food, as in gastric irritation and insufficiency. There may also be a combination of pain in the chest, and in the epigastrium or back; or no pain may be complained of, although localised tenderness is present, and the history of hæmatemesis shows the case to be one of gastric ulcer.

Vomiting.—Vomiting is a symptom frequently present and sometimes absent in cases of gastric ulcer. It may be absent when the history of hæmatemesis shows the case to be one of ulcer and when epigastric pain and tenderness is actually present. If, however, the pain is severe, vomiting is rarely absent. Vomiting usually comes on after a meal, at varying intervals; sometimes directly, sometimes in half an hour, at other times in two hours or even later. In the latter case it is associated with great delay of food in the organ. In vomiting coming on soon after food it is due to a state of irritation of the ulcer. It is frequently observed that the patient gives a history of occasional vomiting after food, and that through neglect or injudicious feeding the vomiting increases until everything is rejected from the stomach as soon as it is swallowed. These cases of continuous vomiting and retching are of great importance, and are due to an excessive irritability of the stomach set up by neglect of treatment or sometimes by a single injudicious meal. The character of the vomited matters has been previously described (p. 421).

Hæmatemesis and Melæna.—The occurrence and symptoms of hæmatemesis and melæna have been described in Chapter XIII. In hæmatemesis due to gastric ulcer the bleeding may be severe or slight; it may recur at intervals, perhaps of years, and cases are not rare where it has occurred in girlhood and recurred in middle age. Its onset is preceded by no warning, and a large quantity of blood may be brought up at once or in two or three vomitings in the course of a week. It comes on usually after a meal, either directly or after a short interval, and it is preceded usually by the symptoms of pain and tenderness previously described; but it may be the first notable symptom in gastric ulcer. The percentage of cases in gastric ulcer in which hæmatemesis occurs is said to be from 30 to 40, but it is much higher than this. In 171 cases, collected from the records of University College Hospital, hæmatemesis occurred in 144, a percentage of about 84. This figure is perhaps too high, since a large number of cases are admitted into hospital only when they are the subjects of an attack of hæmatemesis. Even making allowance for this, the percentage of cases in which the symptom occurs cannot fall short of 80.

The profuseness of the hæmorrhage does not depend on the size of the ulcer, although to some extent no doubt it depends on the situation. A profuse and fatal hæmatemesis is usually due to the opening of a large artery, such as a branch of the splenic, of the pyloric, or of the coronary artery. Melæna is a symptom which, as has been seen, occurs when hæmatemesis is present, but in slight bleedings and when the ulcer is near the pyloric orifice melæna may occur without hæmatemesis, as in duodenal ulcer. In the 171 cases in University College Hospital already quoted, melæna was observed in 19 cases, a percentage of 11, and in all these cases hæmatemesis was also present.

The occurrence of hæmatemesis may be in relation to a meal, or there may be no relation between the bleeding and the taking of food. In these latter cases vomiting may not occur, and all the blood may be found in the stools (Brinton), a fact which emphasises the importance of the systematic examination of the stools in cases of suspected or evident gastric ulcer.

Flatulence is a symptom not infrequently observed in gastric ulcer. It is not due to bacterial fermentation of the food, but to one or other of the causes previously discussed (p. 161). The accumulation of gas may cause great distress by distending the stomach, and is one of the accessory causes of perforation. It tends also to produce dilatation of the organ (see Dilatation, Chapter XIV.).

The *appetite* does not vary considerably in ulcer of the stomach. It is commonly described as good, but the patients are afraid to eat owing to the pain caused by food. There may even be an increase of appetite. In long-continued ulcer, however, the appetite fails, and there may be complete anorexia. The appetite may also be affected by a general disease of the body associated with the gastric ulcer (p. 169).

The *tongue* varies. It is usually clean, and not infrequently the broad, pale, flabby, tooth-indented tongue of anæmia is seen.

The *bowels* are usually constipated, hard motions being passed. Diarrhœa is not a common symptom. It may be present when much blood is passed in the stools, but is more common in duodenal than in gastric ulcer. In either case it is never severe.

The *urine* presents no great variation from the normal, except that albumoses may be present (p. 119).

The symptoms of gastric ulcer may be modified by the presence of those of gastric irritation or of gastric insufficiency previously described (Chapter VII.). Catarrh may develop in the later stages of the disease, and is usually chronic; it is shown by the presence of mucus in the vomit, which is alkaline or contains little or no free hydrochloric acid. Not uncommonly the presence of catarrh is only diagnosed at the post-mortem examination. Moderate dilatation of the organ due to weakness of the muscular coat is not uncommon in cases of gastric ulcer, the origin of the muscular weakness being chiefly the presence of anæmia.

Physical Examination.—Physical examination of the abdomen in the typical cases of ulcer under consideration shows the presence of local tenderness in the epigastrium and back, and a moderate degree of dilatation of the organ. There is no tumour except in the rare cases of fibroid stenosis of the pylorus. The regions of the abdomen other than the epigastric are usually quite normal, although hardened fæces may be felt in the cæcum or the sigmoid flexure.

Irregular or Latent Symptoms.—Ulcer of the stomach may be present and the typical symptoms just described absent. These cases occur both in young women and in middle aged patients of both sexes. The symptoms may be simply those of a period of "dyspepsia," and consist of a sense of weight after taking food and pain in the chest, followed at varying intervals by hæmatemesis, which is the first sign that any other disease than that of functional disorder of the stomach is present. In other cases the first sign is that of perforation, and although such patients may exhibit signs of ill-health previous to the perforation, no symptoms referable to gastric ulcer are discoverable. In other cases, again, there are symptoms of functional disorder of the stomach which may be succeeded by attacks of severe vomiting and retching, in which very acid vomit is brought up. Hæmatemesis or perforation may not ensue in these cases, which are only suspected to be gastric ulcer. In still other cases there are no symptoms whatever during life suggesting gastric ulcer, which is first discovered on a post-mortem examination. Some cases of perforation and of peritonitis belong to this class, as well as cases in which the stomach symptoms are overshadowed by those of a severe co-existing disease, such as bronchitis, tuberculosis, and renal disease. In suspected cases of gastric ulcer where hæmatemesis is absent, the diagnosis rests chiefly on the presence of a localised pain after food or of localised tenderness in the epigastrium or back, in the regions already described.

Effect on Nutrition.—In the early stages of gastric ulcer, patients do not waste; the digestion is good, and sufficient food is absorbed to maintain the nutrition of the organism. On the other hand, if there is much vomiting, wasting is observed, and it may be well marked. Attacks of hæmatemesis are also followed by wasting, which is chiefly due to the enforced starvation of the patient. In ulcer of long duration, on the other hand, the wasting is frequently very great; the patients being emaciated, anæmic, and very weak. This condition is brought about either by deficient digestion of the food owing to the diminution of the secretion of the gastric juice or to the presence of chronic gastric catarrh, or by the supervention of one of the complications of ulcer, viz. simple dilatation of the organ, stenosis of the pylorus, or contraction due to stenosis of the cardiac orifice.

Course and Duration of Ulcer.—According to Cruveilhier, 80 per cent of the cases get well, but this is probably too high an estimate of recovery. It is a disease which is amenable to treatment and is cured by treatment, relapses being, in the large number of cases, due to injudicious feeding, to some other irregularity tending to increase or to aggravate ulceration, and to the fact that it is difficult to persuade patients to be careful with their diet when they feel well. It is a disease in which there are remissions and intermissions: that is, there are periods when the typical symptoms above described are present; then these cease entirely and the patient has a period—varying from months to years—of complete or partial relief from the symptoms, which then reappear, sometimes suddenly—with hæmatemesis due to some dietetic or other irregularity. Relapses which occur at long intervals may be due either to the opening up of an old ulcer, or to the formation of a new one; sometimes the new ulcer is acute, perforation ensuing and rapid death. The duration therefore of an ulcer cannot be stated to be of any definite length: the patient may be ill only a few weeks and then the ulcer perforates, causing death; or symptoms may be present for months, even for years, and then recovery takes place. It is in recovery following old ulcers that the complications shown in distortion of the stomach are most common.

Prognosis.—The prognosis of ulcer, speaking generally, is good. Not only do many cases recover, but healing is so greatly promoted by treatment, that in the majority of cases it may be said that the prognosis depends on the proper treatment being adopted, and many cases recover without any deformity of the organ produced by cicatrisation of the ulcer. Accidents may, however, happen at any time, viz. perforation or a fatal hæmatemesis. The only means of guarding against perforation and hæmatemesis is by treatment. Both perforation and hæmatemesis may come on in patients who have not previously experienced any severe stomach symptoms, and so for the first time come under medical observation when these accidents have happened. On the other hand, when the patient is under medical supervision, these accidents may be to a great extent prevented by the treatment of the patient. No prognosis can be made from the size, depth, or position of the ulcer, for at present there are no symptoms or physical signs enabling the diagnosis of these points to be made.

In determining the prognosis the general condition of the patient must be taken into account, that is, the association of other diseases with the ulcer of the stomach; chlorosis, cardiac disease, renal disease, and tuberculosis. Chlorosis does not seriously affect the prognosis inasmuch as it is capable of being relieved by treatment. Both in cardiac—that is, mitral valvular disease—and in chronic renal disease the tendency is to hæmorrhage not only from the stomach, but from other parts; and in some cases of mitral disease associated with chronic ulcer, hæmatemesis is a well-marked symptom. In uncompensated mitral disease, when gastric ulcer exists, the condition may be serious, but the prognosis here depends on the degree with which the compensation of the heart can be remedied by digitalis or other remedies. The prognosis of gastric ulcer in renal disease is serious, but not from the presence of the ulcer itself, but from the dangers of the renal disease. In tuberculosis, the lung symptoms not unfrequently obscure to a greater or less extent those of gastric ulcer, which is then found for the first time on a post-mortem examination. In cases of slight tuberculosis the gastric ulcer may be shown by definite symptoms; it is a serious complication of such cases, although not necessarily, or even commonly, fatal. In cases of old ulcers where there is great anæmia, wasting, and even emaciation, a guarded prognosis must be given. One or two slight attacks of hæmatemesis may be in these cases fatal, and the deficiency in the functions of the stomach are only partly, and not completely, capable of being remedied by treatment.

DIAGNOSIS.

In the majority of instances, the diagnosis of gastric ulcer is easy. The association of the pain directly after meals with local tenderness and with vomiting, and the occurrence of hæmatemesis with or without melæna, is sufficient for the diagnosis of the condition. This combination of symptoms is not always present. Pain after food is present in other diseases of the stomach besides gastric ulcer, for example in "nervous dyspepsia," in gastric catarrh, etc.; and there are other causes of hæmatemesis than gastric ulcer, while hæmatemesis is not always present in gastric ulcer. The diagnosis therefore has to be considered from several points of view—as regards the age and sex of the patient in which the symptoms occur, as regards the presence or absence of hæmatemesis, as regards the absence of all the typical symptoms pointing to ulcer.

In a young woman the subject of chlorosis, or even without chlorosis, where there is no renal disease and no cardiac disease, the presence of localised epigastric and dorsal pain, with localised tenderness in these regions, coming on after food and accompanied by vomiting, giving relief to the pain, is sufficient for the diagnosis of gastric ulcer, whether hæmatemesis be present or not. On the other hand, with such patients hæmatemesis may be the first symptom noticed, and its occurrence, even with slight dyspeptic symptoms, is sufficient for diagnosis.

In middle age — both in women and in men — the significance of the local pain and tenderness after food is as great as in young adults, but the diagnostic significance of hæmatemesis, when these special symptoms are absent, is not so great, inasmuch as in middle age other diseases are common which may produce hæmatemesis—such as mitral valvular disease, which, arising in young adult life and becoming compensated, is liable to become uncompensated in middle life, and chronic renal disease which frequently develops towards middle life. It has been previously pointed out that although hæmatemesis may be caused by these diseases, yet gastric ulcer may be associated with them, and produce hæmatemesis. The question of the diagnosis of hæmatemesis as to whether it is due to stomach bleeding or not has already been discussed (Chapter XIII.), and need not again be referred to. When hæmatemesis is absent the diagnosis of the gastric ulcer rests on the character of the pain and tenderness almost solely, but pain may be absent, and the tenderness be brought out only by moderately deep palpation in the epigastrium. In not a few cases, the diagnosis of ulcer is made after perforation has occurred (see Perforation, p. 442).

Ulcer of the stomach must be diagnosed from the following conditions:—Gastric catarrh, nervous dyspepsia, gastralgia, carcinoma ventriculi, cholelithiasis, duodenal ulcer.

1. *Gastric Catarrh.*—It is only in rare cases that a case of gastric catarrh could be mistaken for one of ulcer. In catarrh there is pain after food, but the pain differs from that of ulcer in the fact that although it may be epigastric, yet it is diffuse, and never presents the localised character of the pain and tenderness in ulcer. Moreover, there is this important difference between the tenderness and pain in gastric ulcer, and that in catarrh. It has been shown previously that in subacute catarrh the whole of the stomach region may be painful and tender, and that in catarrh in the stage of recovery or in chronic catarrh, there may be one or two tender areas in

the epigastrium, but that these are not constant, and rapidly disappear, in many instances appearing perhaps over another area of the stomach. In ulcer, on the other hand, the pain and tenderness is always localised to one spot, and it is remarkable how long—it may be months—this particular spot may be painful and tender after food. The character of the vomiting in catarrh does not differ very much from that in ulcer, and in subacute catarrh it has the same relation to food as in ulcer. The vomit in catarrh differs from that of ulcer in the fact that it contains mucus, and never contains an excess of hydrochloric acid as in some cases of ulcer; the motions also may contain mucus. Hæmatemesis may be present in both diseases, but in gastric catarrh it is usually absent, and when present it exists only in a small amount. Profuse hæmatemesis is never due to gastric catarrh. In subacute catarrh and in catarrh generally there is a greater tendency to dilatation of the organ than in ulcer.

2. *Nervous Dyspepsia and Gastralgia.*—Cases of nervous dyspepsia and gastralgia frequently present great difficulty in their diagnosis from chronic ulcer. This generally arises from the great pain present, and in many cases its association with repeated vomiting. The pain in gastralgia varies greatly, but it is usually sharp and shooting—it comes on when the stomach is empty, and rarely is it increased by the ingestion of food, being usually indeed relieved by eating. It differs again from the pain of chronic ulcer by being relieved frequently by alcoholic drinks, the pain of ulcer being usually increased; and it is relieved as a rule by pressure. Nervous dyspepsia and gastralgia are associated with those conditions of the nervous system which have been previously discussed, viz. hysteria, hypochondriasis, and neurasthenia (p. 207).

Some difficulty will arise in those cases of nervous dyspepsia where epigastric distress is associated with repeated vomiting, and where ulcer might be suspected. Besides the association of the stomach condition with general nervous symptoms, the absence of hæmatemesis and melæna is an aid to diagnosis. Moreover, when placed on strict regimen such patients do not improve in ten to fourteen days, as in cases of ulcer (Leube).

3. *Carcinoma Ventriculi.*—As a rule there is no difficulty in distinguishing carcinoma ventriculi from ulcer. The age of the patient may be distinctive; carcinoma appearing in middle age or just beyond it. The pain is again characteristic—it is not like that of ulcer, so directly associated with the ingestion of food, and is more or less continuous or appears in severe paroxysms. The vomiting is not related to the pain, and is usually that associated with dilated stomach, and the vomited matters show a diminution in the amount of hydrochloric acid, and the products of bacterial fermentation are found (Chapter XIV.). Hæmatemesis is not so frequent or profuse in cancer as in ulcer, and is usually shown in "coffee-grounds" vomiting. The presence of a tumour at the pylorus or near the pylorus is distinctive of carcinoma except in those rare cases in which there is a fibroid stricture of the pylorus (see Cancer, p. 492). Cancer is a progressive disease; ulcer, a chronic disease with intervals of quietude.

4. *Gallstones—Cholelithiasis.*—In the majority of cases ulcer of the stomach cannot be mistaken for gallstones; inasmuch as, although biliary colic usually supervenes after a meal, yet the pain is not situated in the stomach region, but to the right of it, and it is extremely severe, extending all over the right hypochondrium and round the abdomen. It is only in the rare cases of ulcer in which the pain is so severe that the patient has to roll on the floor to try and obtain relief, that the pain of biliary colic might be mistaken for that of ulcer. In such cases the history of the patient may be distinctive; the localised pain and tenderness, and the history of hæmatemesis would point to ulcer. The history of long intervals between the attacks of pain is in favour of biliary colic, as well as the occurrence of jaundice and a tender liver after the attack.

5. *Duodenal Ulcer.*—See p. 458.

Diagnosis of the Seat of the Ulcer.—The diagnosis of the seat of the ulcer is impossible in the majority of cases. The position can only be suspected, and no reliance can be placed on the directions which have been given for the diagnosis of the position of the ulcer. Thus an ulcer of the posterior surface is said to be indicated by pain in the back more than in the epigastrium, and in these cases pressure on the epigastrium will cause pain in the back. An ulcer on the anterior surface is said to give great tenderness to the right or left of the middle line with less tenderness at the back, and it is said that the pain after meals is diminished by the patient lying on the left side. An ulcer at the pyloric orifice is said to be distinguished by the pain coming on some time after food, that is, when the contents of the stomach are expelled into the duodenum, and by the fact that the pain is relieved by lying on the left side. But there is not sufficient evidence to go upon to decide whether the diagnosis of the seat of the ulcer is possible from these statements, and it must be remembered that although diagnosis of the seat of the ulcer is important from the point of view of prognosis and of surgical procedure, yet ulcer of the stomach is not always single, and cases are not infrequent where there is an ulcer situated on the posterior surface—the most favourable position—and another on the anterior surface, the most dangerous position.

For the diagnosis of perforation and other complications of ulcer, see p. 440 *et seq.*

TREATMENT.

The two objects to be borne in mind in the treatment of ulcer in the stomach are:—

1. To promote the healing of the ulcer.
2. To relieve the symptoms of the disease.

1. The healing of the ulcer is promoted by means of rest. By giving rest to the stomach, not only is the healing of the ulcer promoted, but the dangers of hæmatemesis and of perforation are averted; and it is in devising means of giving physiological rest to the stomach, while enabling the patient to assimilate sufficient food for the needs of the economy, that the treatment of ulcer consists.

Physiological rest is given to the stomach in the following ways:—

(*a*) *By bodily rest.*—Rest in bed or in the recumbent position is essential to the treatment of cases of ulcer with active symptoms.

(β) By *means of food*, which in its solid form irritates the ulcer and prevents healing, and by exciting peristaltic movements of the organ and the secretion of gastric juice (chiefly the hydrochloric acid) is the direct cause of preventing the healing of the sore. Complete physiological rest is given by cutting off the administration of food by the mouth, and adopting feeding by the rectum; partial physiological rest is given by adopting a liquid diet, one readily digested and non-irritating.

(γ) By preventing gaseous distension of the stomach, whereby the ulcer is stretched and its healing delayed.

(δ) By treating the general condition of the body—chiefly anæmia—which delays the healing of the ulcer.

The chief conditions which prevent the healing of the ulcer of the stomach are injudicious dieting and the presence of a serious general disease.

2. Treatment must also be adopted to relieve the symptoms of the disease, viz. pain, vomiting, hyperacidity, flatulence, constipation, and, lastly, the occurrence of bleeding as shown in hæmatemesis and melæna. The actual treatment to be adopted is both medicinal and dietetic.

Cases of ulcer of the stomach may first come under observation in different stages of the disease.

1. They may be only suspected cases, *i.e.* those in which hæmatemesis has not occurred.

2. There may have been a recent bleeding from the stomach, from the effects of which the patient is still suffering; or the bleeding may occur while the patient is under observation.

3. There may be a history of hæmatemesis some time previously, the effects of the loss of blood having passed off, but there are still symptoms of ulcer, as shown in pain, tenderness, and vomiting; and individual cases may show greater or less prominence of one or other of these symptoms.

4. The case may be of long standing with intervals of quietude, and with attacks of hæmatemesis; and the patient

may show the signs of cachexia and of gastric insufficiency with dilatation of the stomach.

The treatment of cases in which there has been recent hæmorrhage has already been considered in Chapter XIII. p. 377.

The treatment of long-standing cases is that of gastric insufficiency, which has already been considered in Chapter X. p. 295, and Chapter XI. p. 334; or that of dilatation, which has been considered in Chapter XI. p. 347, and Chapter XIV. p. 391.

The remaining cases for consideration are the suspected cases, and those in which the diagnosis is certain and there are actual symptoms of ulcer, but there are none of the effects of long-continued ulceration of the organ.

The diet which is to be adopted in these cases has already been discussed (Chapter XI. p. 349). The choice is between feeding by the mouth and rectal feeding. And in feeding by the mouth the diet is to be prescribed on the lines discussed in Chapter XI. p. 340 *et seq.* In changing from the liquid diet to the solid one, great difficulties will be experienced, and the change must be effected on the lines previously laid down (Chapter XI. p. 322 *et seq.*), viz. in the graduated feeding adopted for the cure of gastric irritation and chronic gastric catarrh. The principles of dietetics in chronic ulcer do not differ from those in other gastric affections, and have already been so fully discussed that they need no repetition here.

As regards the medicinal treatment in chronic ulcer, acids are, as a rule, inadmissible, and alkalies are of great service, acting as antacids (Chapter X. p. 291). They may be with benefit combined with sedatives for the relief of pain and vomiting. Sedatives may be given by themselves for the relief of pain and vomiting (Chapter X. p. 300).

For the treatment of flatulence and gaseous distension of the stomach, see Chapter X. p. 294. For the treatment of constipation, Chapter X. p. 309. It may be added that in chronic ulcer all violent purgatives are to be avoided.

Results of Treatment.—By the adoption of the proper treatment, patients with ulcer of the stomach greatly improve, whether there has been a recent hæmatemesis or not. By continued rest to the body and mind, by the careful administration of food by the mouth or by rectal feeding, and by the judicious administration of remedies for the relief of the symptoms, patients make good progress in fourteen days or a month. Any relaxation of the treatment must be allowed only after careful consideration of its effects in relieving the symptoms. A time comes when the liquid diet by mouth causes no epigastric distress, and when, by the slow addition of carbohydrates or digestible animal food articles to the diet, no pain is experienced after food. As long as there is local tenderness in the epigastrium the patient must be carefully dieted; any increase in the food which causes pain being at once withdrawn from the dietary. The patient may gradually be able to take an ordinary diet of digestible food articles (p. 327). But this is the period of danger. The ulcer perhaps has healed, but the cicatrised tissue is soft, and dietetic indiscretions in the form of excess of food, of indigestible food, or of irritating food accessories may bring on a return of the symptoms. These indiscretions indeed are the cause of relapses; they are the cause of the failure of the after-treatment of ulcer. Patients who have had an ulcer and who feel well lose their caution and consume the diet of a healthy person, whereas for a long time (it may be a year or more) after the active symptoms of ulcer have subsided, the patient ought to consider himself a dietetic invalid.

Complications of Ulcer.

The two dangers of ulcer to the stomach are hæmatemesis and perforation; they are the commonest causes of death, in addition to exhaustion produced by an ulcer of long standing. It is impossible exactly to estimate the proportion of cases which die from each of these causes, but in statistics it seems to be shown that the females are more subject to death from perforation, and males to death from hæmorrhage.

Hæmatemesis and melæna have already been considered (Chapter XIII.). The following complications of ulcer remain for discussion: dilatation of the stomach, hour-glass contraction of the stomach, and perforation.

Dilatation.—Simple dilatation occurs in a certain number of cases, and may be of two kinds, viz. a temporary dilatation occurring in the early stage of the ulcer and due to causes previously mentioned, or a permanent dilatation which occurs more frequently in patients in middle and old age. Habershon[1] found that in sixty cases of ulcer there were eighteen cases of dilatation of the stomach, and that the period at which these occurred was at an average age of fifty-two ; this includes both simple dilatation and dilatation due to stenosis. Permanent simple dilatation is not an uncommon consequence of ulcer, and is more frequent than that due to stenosis of the pylorus. The symptoms have already been described under the heading of Dilatation of the Stomach (Chapter XIV.). In the history there is usually clear evidence of the existence of an ulcer from the occurrence of pain and vomiting after food, with attacks of hæmatemesis. The diagnosis and treatment have also been considered, and the diagnosis of stenosis as the cause of dilatation will be considered under the head of Cancer (Chapter XVI. p. 494).

2. *Hour-glass Contraction of the Stomach.*—Hour-glass contraction of the stomach may be either congenital, or it may be due to the cicatrisation of a large ulcer in the mid-region or the pyloric region of the stomach. It is a condition not usually diagnosed during life, and it is usually discovered only at the post-mortem examination. There may be evidences of dilatation of the organ, and the only sign which points to the presence of hour-glass contraction of the stomach is one described by Jaworski under the name of *ectasia ventriculi paradoxa.* Simply stated, this means that there may be evidences of the presence of a large amount of fluid in the stomach on succussion ; or a large amount of fluid may be passed into the stomach by the tube, and yet but little can be regained by means of the pump or aspirator ; that is, the fluid passes into the farther compartment of the stomach, and cannot be regained. There is no treatment special to hour-glass contraction of the stomach except that for other forms of dilatation.

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[1] S. H. Habershon, M.D., "The Prognosis of Simple Gastric Ulcer," *St. Barth. Hosp. Rep.*, London, vol. xxvii.

Dilatation of the pyloric region into a pouch not infrequently results from ulcer. This dilated portion of the organ may sometimes be felt during life, occupying the right part of the stomach region. The rounded mass gives a tympanitic note on percussion.

3. *Perforation.*—Perforation is a frequent cause of death, and is the event most to be dreaded when a gastric ulcer is present. According to Brinton, in 234 cases of death from perforation, 160 were females and 74 were males; in statistics quoted by Habershon, death from hæmorrhage took place in 185 cases, of which 108 were in men and 77 in women. Dilatation, it may be mentioned, according to Habershon, is also more frequent in males than in females.

The frequency of perforation has been estimated by Brinton as 13½ per cent of all cases, the calculation being made from 234 fatal cases; according to Habershon perforation occurs in 18 per cent of all cases. It is most frequent in young females, and is more or less likely to occur according as the ulcer is situated towards the anterior surface of the organ where there is no protecting solid organ. In ulcers situated on the posterior surface, near the pancreas, spleen, or liver, perforation may have several effects. The walls of the stomach may be completely adherent to the solid organ, and, the base of the ulcer being perforated, a large abscess may subsequently be formed in the liver, pancreas, or spleen. In other cases the perforation is into the peritoneal cavity. According to the position of the ulcer perforation may have one or two effects; the contents of the stomach may be extruded into the general peritoneal cavity, or they may be shut into the upper peritoneal cavity, usually by adhesions. In the first case rapid death may occur before peritonitis is developed; general peritonitis, however, being the usual consequence of the accident. In the second case an abscess is formed (subphrenic abscess) either on the left or the right side of the upper part of the abdomen.

Anatomically, therefore, perforation of the ulcer may occur without serious effects, the base of the ulcer being formed by a solid organ, and the stomach contents prevented from escaping by adhesions round the base of the ulcer. Clinically the term perforation is best limited to those cases in which the stomach contents escape into the peritoneal cavity.

Perforation into the General Peritoneal Cavity.—This usually occurs from ulcers on the anterior surface of the stomach, nearly 80 per cent of which are said to perforate. Death may occur in a few hours from shock without the development of general peritonitis. In other cases the symptoms may be divided into two stages, in the first of which there are evidences of the sudden rupture of a hollow viscus, and in the second the evidences of general peritonitis. The rupture usually occurs without warning, taking place after a meal, not uncommonly of indigestible food, or after some sudden exertion, perhaps on a full stomach; or it may occur in a fit of sneezing or coughing and in vomiting. Perforation has also been known to occur after the administration of strong remedies, *e.g.* purgatives. There is acute pain referred to the upper part of the abdomen, sometimes doubling the patient up, with faintness and vomiting. The pulse is extremely rapid; there may be dyspnœa and the features soon become pinched, drawn, and haggard. The abdominal pain is first localised in the upper part of the abdomen, and then becomes more diffused. A physical examination made at this stage frequently reveals some definite signs. The contents of the stomach, which consist of acid fluid and food-remains with gas, are extruded into the general peritoneal cavity. The escaped stomach contents may either be retained in the upper part of the peritoneal cavity, *i.e.* between the diaphragm and the upper abdominal organs (liver, stomach, and spleen), or they may pass to the lower part of the cavity. The position which the escaped contents occupy is determined by two causes: the position of the ulcer and the presence of adhesions. In perforation of ulcers of the anterior surface, when there are no adhesions, the stomach contents enter the lower part of the peritoneal cavity, as in duodenal ulcer. But when there are adhesions and in perforation of ulcers of the posterior wall, the stomach contents are often retained in the upper part of the abdomen. In this case the physical signs are the same as those described under subphrenic abscess (p. 446).

The presence of fluid and gas in the lower part of the peritoneal cavity may not be discoverable owing to the rigidity of the abdominal muscles, but it may be seen that the abdomen generally is moderately distended, the degree of distension depending on the amount of gas and liquid in the stomach at the time of perforation. Palpation may show tenderness over the stomach region, not usually localised, although it may be most marked at the seat of the perforated ulcer. Percussion may demonstrate the presence of gas in the abdominal cavity, and liquid in the flanks and iliac regions, but if the amount of liquid is small it may be all collected into the pelvic cavity, and thus be undiscoverable by a physical examination of the surface of the abdomen. In a previously healthy individual, for example in a young woman who has had no previous serious illness, these symptoms and signs are indicative of perforation.

The patient is, however, as a rule seen in the second stage, when peritonitis has supervened. In this stage there is fever which is not as a rule high, ranging from 100° to 102° F.: it is continuous in the early stage, but, later on, it becomes irregular. The patient not unfrequently lies in bed on the back, with the knees drawn up, with an expression of pain in the face, and a drawn countenance. The pulse is rapid, small, and there is a moderate increase of arterial tension. Vomiting and retching may be frequent and hiccough may supervene. A physical examination in this case may reveal a small amount of gas and liquid in the peritoneal cavity as in the early stage, but extreme rigidity and distension of the abdomen may prevent any other sign being discovered except extreme tenderness, chiefly over the lower two-thirds of the abdomen. The presence or absence of abdominal respiration in the first or second stage is of some importance. In the second stage, that of peritonitis, it is usually absent; in the first stage it may be present or absent, and at the onset of perforation it is usually absent.

Diagnosis.—The diagnosis of rupture of gastric ulcer from rupture of any other part of the intestinal tract is in many cases of extreme difficulty, and even at the time when an operation is performed for relieving the patient the diagnosis may not be made. Symptoms of shock followed by those of general peritonitis are common to sudden rupture of any part of the gastro-intestinal tract. In enteric fever the diagnosis is as a rule not difficult, owing to the history of the preceding acute illness and the presence of high fever and diarrhœa; as a rule diarrhœa is absent in cases of gastric ulcer, and it may be absent in enteric fever. In cases of rupture of a duodenal ulcer no differential diagnosis is possible unless at the operation. In cases of rupture of other parts of the intestine the points to be looked to are the occurrence of the first symptoms, pain, etc., as referable to the upper part of the abdomen in gastric ulcer; but in some cases where the history of the illness has to be depended on, this point cannot be clearly made out. In cases of perforation of the large gut, due to malignant disease, there is as a rule no difficulty in the diagnosis; the history, the wasting, the cachexia, and the presence of a tumour, with the signs of chronic intestinal obstruction, suffice to show the nature of the case. At the operation undertaken to relieve this condition the diagnosis that the rupture has occurred in the stomach or first part of the duodenum may be made by finding an acid fluid in the peritoneal cavity, and especially a fluid containing free hydrochloric acid. The tests for this are easily performed (Chapter V.), and if free hydrochloric acid is found, the liquid must have come from the stomach or first part of the duodenum. Hydrochloric acid soon disappears from the effused liquid when peritonitis supervenes or when decomposition takes place, so that its presence will only be shown in early cases of perforation.

Prognosis.—Almost invariably, if untreated, cases of perforation of a gastric ulcer into the general peritoneal cavity are fatal, either immediately from shock or after a few days from the effects of general peritonitis. Cases have been recorded, however, in which spontaneous recovery has taken place, but these are extremely rare, rarer perhaps than in cases of typhoid fever where there has been perforation.

Treatment.—The only treatment that can be suggested as likely to save the life of the patient is surgical; that is, the performance of an abdominal section. The two objects of such an operation are the thorough cleansing of the peritoneal cavity, and, if possible, the closure of the opening in the ulcer. The decision as to what has exactly to be done depends partly on the accessibility of the ulcer and partly on the adhesions round the ulcer. The ulcer may be inaccessible or may be surrounded by so many adhesions that it is unadvisable to interfere with it. The patient is in great danger from two causes. If the stomach contents are allowed to remain in the peritoneal cavity, they will almost invariably cause death by peritonitis and by putrefactive decomposition. This is the immediate danger in which a patient lies, and is obviated by the thorough cleansing of the peritoneal cavity. The second danger is the rupture in the ulcer. If this be not treated a further extrusion of the stomach contents may occur, causing peritonitis and death after the peritoneum has been cleansed. There are cases in which the rupture has been closed by operation, and it has been found subsequently that the same ulcer has perforated in another place, causing the death of the patient. If it is a question of excising the ulcer, there will be no difficulty regarding this if the ulcer is small and on the anterior surface, but it is impossible to determine the size of the ulcer from the size of the perforation and the occurrence of the horseshoe-shaped ulcer must be remembered, that is the long oval ulcer which extends from the posterior surface across the lesser curve to the anterior surface, and not unfrequently perforates on the anterior surface. The subsequent treatment of cases after operations must be conducted on general lines, complete rest being given to the stomach by rectal feeding.

Subphrenic abscess is the second result of a perforation of the gastric or duodenal ulcer. It is the formation of an abscess below the diaphragm shut in by adhesions between the stomach and the surrounding organs, and is produced when the escaped stomach contents are retained in the upper part of the abdominal cavity (p. 443), *i.e.* between the diaphragm above and the liver, stomach, and spleen below. It may occur from perforation of an ulcer in any part of the duodenum and stomach except the lower part of the anterior wall. When the ulcer is situated to the right, that is near the pylorus, a subphrenic abscess is formed on the right side; when the perforating ulcer is on the posterior wall of the stomach, on the anterior wall or near the cardiac end, an abscess is formed on the left side of the body. In ten cases published by Drs. Penrose and Dickinson[1] of left subphrenic abscess, the condition was due to perforation of an ulcer on the posterior surface in four cases, and on the anterior surface in six, two of the latter being near the cardiac end. In 19 of the cases collected by Maydl,[2] the perforating ulcer was found in 12 cases on the small curve or near the cardia; in 6 cases on the posterior wall, and in 1 on the anterior wall.

Subphrenic abscess is not always due to perforating ulcer of the stomach or duodenum. According to Nowack,[3] who collected 78 cases, only 41 per cent were due to this cause, and 4 per cent were due to perforation in cancer of the stomach or œsophagus. The other cases were chiefly due to echinococcus of the liver, typhlitis, injury, gallstones, splenic abscess, or perinephritic abscess. Some of these conditions produce an abscess in the cellular tissue beneath the peritoneum, and are thus extra-peritoneal, and this occurs in the extension upwards of abscesses round the cæcum or kidney or in those following an injury. In the majority of cases, however, the abscess is intra-peritoneal, and this occurs in rupture of a gastric or duodenal ulcer, and in the rupture of a liver or splenic abscess into the peritoneal space beneath the diaphragm. It is only the intra-peritoneal variety which will be discussed here.

Morbid Anatomy.—The morbid anatomy of intra-peritoneal subphrenic abscess is best explained by reference to the accompanying figure (Fig. 49) taken from Leyden's original paper.[4] This was a case of right-sided subphrenic abscess due to the perforation of a duodenal ulcer. The arch of the diaphragm is seen to be pressed upwards as far as the third intercostal space on the right side by a large cavity which compresses the lower lobe of the right lung, presses downwards the liver and displaces the heart slightly towards the left. The left wall of the abscess is formed by the falciform ligament of the liver, its lower wall by the liver; on the right it is bounded by the thorax, and above by the diaphragm. When the subphrenic abscess exists on the left side of the body its boundaries are formed as follows: above, by the diaphragm; below, by the left lobe of the liver and the anterior surface of the stomach; in front, by the abdominal wall united by adhesions to the anterior surface of the stomach; on the right side by the falciform ligament, and on the left by adhesions between the cardiac end of the stomach, the spleen, and the diaphragm. Although originally right- or left-sided, a subphrenic abscess may infect the opposite side, and thus occupy all the space beneath the diaphragm. In this case, the falciform ligament is sometimes perforated, and sometimes intact.

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[1] *Trans. Clin. Soc.*, London, 1893, vol. xxvi. p. 72.

[2] Carl Maydl, "Ueber subphrenische Abscesse," Wien, 1894.

[3] E. Nowack, "Die hypophrenische Empyeme," Schmidt's *Jahrb.*, Leipzig, Bd. ccxxxii. pp. 73 and 200.

[4] E. Leyden, "Ueber Pyo-pneumothorax subphrenicus (subphrenische Abscess)," *Ztschr. f. klin. Med.*, Berlin, 1880, Bd. i. p. 320.

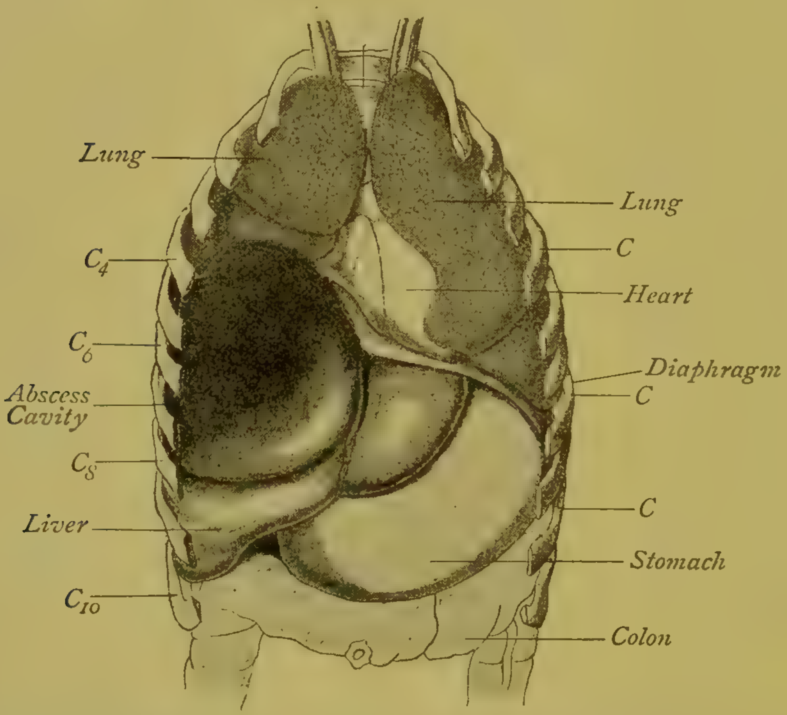

FIG. 49.—Showing the anatomical relations of a right-sided subphrenic abscess, arising from perforation of a duodenal ulcer. The abscess has pushed up the diaphragm on the right side, compressing the lower lobe of the lung, and pushed the liver downwards, and the heart slightly to the left. *C* = ribs. (Leyden.)

The formation of a large abscess in this neighbourhood compresses the lung above it, displaces the heart either away from it or directly upwards, and if on the right side pushes down the liver, even as far as the umbilicus. The contents of the abscess vary somewhat. In all cases they contain pus and gas; in some cases they are sweet, in other cases foul smelling, owing to putrefactive decomposition. The gas in the abscess is derived partly from the gas present in the stomach at the time of perforation, and partly from putrefactive decomposition occurring subsequently in the extruded stomach contents. The remains of food may be found in a recent abscess.

The perforation in the stomach shows various appearances. It is usually patent, but it is sometimes closed, a pucker being left in the ulcer showing where it had perforated.

Effect on the Neighbouring Parts.—An abscess in this situation, and often a foul abscess as it is, will excite neighbouring inflammation, and therefore the usual accompaniment of subphrenic abscess is an affection of the pleura and lung above it. Thus pleurisy, pleurisy with effusion, empyema with sweet or fœtid pus may be present on the same side as the abscess; the lung may be pneumonic, or an abscess may be developed in it and extensive destruction of the lung may occur in consequence, or gangrene may result. Affections of the pleura and lung may occur without a direct connection between the abscess and pleura; in some cases, however, the pleura and abscess communicate through an opening in the diaphragm. Of 45 cases collected by Maydl, the pleura was normal in 11, adherent in 10, and contained a serous fluid in 9, and pus in 15 cases.

Symptoms of Subphrenic Abscess.—The development of a subphrenic abscess is preceded by the symptoms which have been described as occurring in perforation of the stomach or duodenum and in the peritonitis resulting; that is, there is sudden pain followed by fever, tenderness of the abdomen, and the other signs of peritonitis. But there are soon signs of localisation of the inflammation to the upper part of the abdomen; and in the fully developed subphrenic abscess, the symptoms resemble those of pneumothorax; there being fever of varying intensity, usually from about 100° to 102° F., with dyspnœa, but as a rule no cough or expectoration. The general condition of the patient indicates an appearance in many cases of severe illness, and there is bodily weakness with an expression of pain and a drawn countenance. The chief signs of the disease are elicited by physical examination.

Physical Examination.—Reference to the figure already given of the anatomy of subphrenic abscess and to the accompanying figure (Fig. 50) will explain the physical signs of subphrenic abscess.

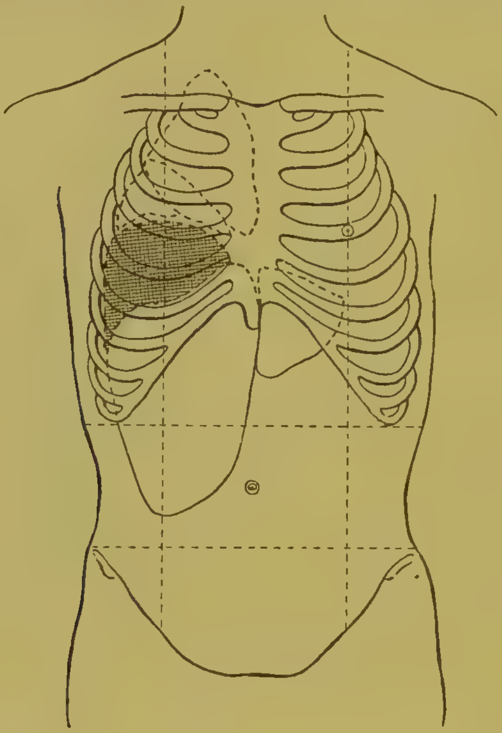

Fig. 50.—Diagram illustrating the physical signs in a case of right-sided subphrenic abscess. The abscess is represented by the dark shading, and over this area in front was obtained a tympanitic note on percussion. Posteriorly over the abscess the note was dull. The limit of tympanitic resonance was sharply marked off above by the lung resonance, and below by the liver dulness. Over the abscess in front distant amphoric breathing was heard and the bell-sound was obtained: both these physical signs ceasing abruptly above, at the limit of the lung, and below at the limit of the liver. The liver was pushed down into the abdomen and the heart towards the left. (Nowack.)

Inspection.—The heart's apex is slightly displaced horizontally away from the disease; in right subphrenic abscess towards the left, and in left subphrenic abscess either upwards or towards the right.

There is but slight bulging of the side, the intercostal spaces being practically normal. The respiratory movements vary somewhat; in some cases there is cessation of the abdominal movements, in others these are normal; but there is deficient movement as regards expansion and elevation of the same side of the thorax as the abscess.

Palpation.—Palpation confirms inspection in the above points, and in some cases a thrill may be elicited over the abscess by a sudden jerking movement given to the abdominal wall. Tenderness may be completely absent. Examination of the abdomen by palpation also reveals the displacement of the liver, the lower edge of which in right subphrenic abscess may be discovered nearly on a level with the umbilicus. In left-sided abscess the spleen is not usually felt, being pushed against the thoracic wall.

Percussion.—Percussion gives very varying signs. Over the lower part of the thorax on the side of the abscess, there is a tympanitic note over an area varying in extent according to the amount of gas contained in the cavity. The upper limit of this area is rounded and sharply marked off from the resonance obtained over the lung. Below, on the right side, the liver dulness over the lower thoracic wall may be completely absent, a tympanitic note only being elicited; and when present this obliteration of the thoracic liver dulness is an important sign of subphrenic abscess. Posteriorly in the thorax there may be dulness when the patient is in the recumbent position; and this dulness shifts its position with alteration of the posture of the patient. On the left side, similar variations in resonance may be discovered, and in front there is not unfrequently tympanitic resonance in the upper epigastric region, and this may be continuous with an area of similar resonance over the cardiac area.

Auscultation.—Commencing from above, vesicular breathing is heard from below the clavicle down to the upper margin of the tympanitic resonance, diminishing in intensity below owing to the collapse of the lung. Over the area of the tympanitic resonance normal breath sounds are quite absent and are replaced by amphoric breathing. Posteriorly this is absent over the area of dulness. Adventitious sounds may be elicited, viz. metallic tinkle by tapping the abdomen near the abscess, or the bell sound by the usual means.

Variations in these physical signs are observed when, in addition to the subphrenic abscess, the pleura or lung above is affected. There may be above the abscess signs of fluid in the pleura, viz. dulness, with absence of breath sounds; or there may be tubular breathing and increased vocal resonance with crepitations, showing consolidation of the lung. In some cases the signs of pneumonia may obscure those due to subphrenic abscess.

On tapping the abscess both gas and fluid are obtained, the latter having the characters previously described (p. 449). If a mercurial manometer be connected with the abscess cavity it will be found that there is an increase of pressure during inspiration, and a decrease during expiration (Pfuhl). This is directly opposite to what occurs when a manometer is introduced into the pleural cavity, and is a distinguishing point between an abscess above and one below the diaphragm. The test, however, does not always succeed.

Diagnosis—1. *As to the Presence of Subphrenic Abscess.*—The chief point in the diagnosis to be settled is whether the cavity is above or below the diaphragm, whether it is a pyo-pneumothorax or a subphrenic abscess. The physical signs are practically the same in pyo-pneumothorax and in subphrenic abscess; that is, they are the signs of a cavity containing fluid and gas. But the subphrenic abscess differs from pneumothorax in producing less displacement of the heart and less bulging of the side affected. These two physical signs, however, differ so much in pneumothorax that no great stress can be laid on them as a distinction between the two diseases. Of more importance are the physical signs of tuberculosis which are present in pneumothorax, when this occurs in either the early or the late stages of the disease; viz. consolidation or excavation at one or other apex.

The history of the case is of great importance. The absence of cough and expectoration and the development of the disease demonstrates its occurrence in the upper part of the abdomen and not in the chest. From other chest conditions subphrenic abscess is readily distinguished by the physical signs which have been dealt with. It must be borne in mind, however, that diseases of the lung and pleura complicate subphrenic abscess; and physical signs may be obtained posteriorly, and above the upper line of tympanitic resonance which indicate an affection of the lung or pleura—pleurisy with effusion, empyema, pneumonia, or pneumonic abscess.

2. *Diagnosis as to the Cause of the Subphrenic Abscess.*—The diagnosis as to whether perforating ulcer of the stomach or duodenum is the cause of the abscess is not always easy to decide, and rests on the previous history of ulcer as regards pain directly after food, vomiting, hæmatemesis, melæna, etc., and on the occurrence of the symptoms of perforation as previously described. The history of ulcer may be absent, but the symtoms of perforation always precede the development of a subphrenic abscess due to ulcer.

The cases due to carcinoma may be diagnosed from the presence of symptoms peculiar to cancer of the œsophagus or stomach (p. 477). It is in the later stages of carcinoma that perforation occurs and it is a rare event.

Abscesses occurring on the right side of the upper part of the abdomen may be in the liver or may be subphrenic and due to typhlitis and perinephritis (when it is extra-peritoneal), or to rupture of a gastric ulcer (when it is intra-peritoneal). On the left side, the abscess may be due to a gastric ulcer or to disease of the colon or kidney. Great difficulty is sometimes experienced in the diagnosis between a collection of pus in the liver and one between the liver and the diaphragm. In the latter case the abscess may contain gas: in the former there is either the history of dysentery preceding the illness or there may be the negative history of a benign tumour (hydatid). The exact nature of some of these cases is, however, unrecognised before an operation. With typhlitic abscess the diagnosis rests on the previous history of disease localised in the right iliac fossa and the presence of actual signs of disease in that situation, viz. induration or abscess. With renal abscess, the diagnosis may be made clear by a history of the symptoms of calculus, and by the presence of pus in the urine.

Course of the Disease.—Subphrenic abscess is a serious condition, which ends fatally either by septic poisoning or more commonly by producing putrid empyema and pneumonic abscess. In some cases the abscess discharges itself through the lung, and although this must be considered the most favourable natural mode of cure of the disease, yet it is one which entails a prolonged illness to the patient, and which sets up destructive changes in the lung. In other cases, the abscess bursts into the general peritoneal cavity, causing death by general peritonitis. The rapidity with which subphrenic abscesses kills varies greatly; it is to some extent dependent on the affections of the lung which complicate it. Of 178 cases collected by Maydl, 98 died without an operation, 6 healed without operation, and of the 74 operated upon, 35 died, and 39 recovered.

Prognosis.—It may therefore be looked upon as practically a fatal disease unless relieved by operation.

Treatment.—This is purely surgical; and the sooner an operation is performed for the proper draining and cleansing of the abscess the more chance has the patient of recovery.

ULCER OF THE DUODENUM.

Definition.—Ulcer of the duodenum is a primary disease of the organ in which there is destruction of the walls of the gut in precisely the same manner as in ulcer of the stomach. It is a simple ulcer, occurring in an acute and chronic form, and its origin is as obscure as that of ulcer of the stomach. Other forms of ulceration occur in the duodenum; hæmorrhagic erosions may be found, and rarely tuberculous, typhoid, and carcinomatous ulcers.

Etiology.—The etiology of the duodenal ulcer, though practically the same as that of ulcer of the stomach, presents some differences worthy of note.

Age.—Whereas ulcer of the stomach is commonest in early adult life, the incidence of duodenal ulcer is later, the average age being thirty-five to forty years, excluding those cases which occur in association with burns, and which are usually met with in children or in young adults.

Sex.—Ulcer of the stomach is more than twice as common in women as it is in men. Duodenal ulcer is three times as common in men as in women, excepting those cases which occur in burns. Out of 17,652 post-mortem examinations which have been recorded during fifty years at Guy's Hospital, Drs. Perry and Shaw found that in 69 cases of duodenal ulcer, 52 occurred in males and 17 in females; excluding burns, 48 occurred in males and 16 in females. Adding these to other cases which they collected from various sources, they found that 109 cases occurred in males and 48 in females; or excluding burns, 100 in males and 30 in females. This is a proportion of about 3 to 1. In the case of burns the relation of sex is reversed; in 27 cases collected by the same observers, 18 occurred in females and 9 in males. This may be perhaps explained by the fact that owing to the clothes of the female, burns are more severe in them than in males.[1]

Relation to other Diseases.—Duodenal ulcer has a special relation to burns and to septicæmia and pyæmia. Although ulcers of the stomach may be found in these conditions, yet it is much rarer than the formation of a duodenal ulcer.

Burns.—The occurrence of duodenal ulcer in burns which was first brought into prominence in a paper by Curling[2] is an undoubted fact, although its frequency has been somewhat exaggerated. The following table shows the statistical relation of duodenal ulceration to cases of fatal burns. It does not take into account those cases in which recovery from a duodenal ulcer occurs in burns, although this is a possible event.

Name of recorder.	Number of cases of burns examined post-mortem.	Number of cases of duodenal ulcer.
Holmes[3]	125	16
Erichsen[4]	94	2
Perry and Shaw[5] . . .	149	5
	368	23

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[1] *Guy's Hosp. Rep.*, 1893, vol. l. "On Diseases of the Duodenum," by E. C. Perry, M.D., and L. E. Shaw, M.D. This is a valuable record of the morbid anatomy of diseases of the duodenum.

[2] *Med.-Chir. Trans.*, London, 1842, vol. xxv. p. 264.

[3] "System of Surgery," vol. i. p. 394, 3rd ed., 1883. Statistics obtained from various sources.

[4] "Science and Art of Surgery," 10th ed., 1895, vol. i. p. 396. Statistics obtained from University College Hospital.

[5] Statistics obtained from Guy's Hospital, *op. cit.*

From this table therefore the percentage of the occurrence of duodenal ulcer in fatal burns is 6·2. The formation of the ulcer is explained most rationally by the occurrence of septic embolism from the sloughing skin; but in addition to the death of the tissue produced by embolism, the action of the gastric juice in digesting the dead tissue must be taken into account. It occurs in the second period of the burn, that of inflammation.

Septicæmia and Pyæmia.—In certain cases of these diseases, duodenal ulcers are found, and there is no doubt that there is distinct relation between them, and that the occurrence of the ulcer is to be explained by the embolism which is a feature of these diseases. Perry and Shaw collected eighteen cases from the Guy's Hospital records of 17,652 post-mortem examinations, and inasmuch as only fifty-two other cases of duodenal ulcer were found, it is evident that there is a proclivity to the formation of duodenal ulcer in cases of septicæmia and pyæmia. To this number they added three other cases from other sources. In the total twenty-one cases, the septic condition was due in ten cases to a sloughing condition of the skin or cellular tissue, and in the other cases to pyæmia, puerperal septicæmia, septic bronchitis, renal abscess, hip-joint disease, and empyema (three cases). Duodenal ulcer may also occur in the course of chronic Bright's disease and in cardiac disease, but there does not appear to be any special relation between the formation of the ulcer and the conditions met with in these diseases. In the great majority of cases duodenal ulcer exists by itself, that is without the presence in the body of any other lesion which would produce death. In some cases it is associated with gastric ulcer; out of 120 cases collected by Perry and Shaw, in fifteen there was an associated gastric ulcer.

Anatomy and Pathology.—These have been already discussed under the heading of gastric ulcer (p. 402 *et seq.*). It is only necessary to add that the ulcer most commonly occurs in the first part of the duodenum, and is usually single, although there may be two or three ulcers. It may occur in the second part of the duodenum, and two ulcers may be found, one in the first part and one in the second. The ulcer not unfrequently forms adhesions to the surrounding parts, in the great majority of cases to the pancreas, but also to the liver, spleen, or gall-bladder.

The results of duodenal ulcer are practically the same as those of gastric ulcer. Perforation, leading to general peritonitis or to subphrenic abscess, may occur; or stenosis of the gut due to the cicatrisation and contraction of a transverse ulcer; or stenosis of the bile-duct due to cicatrisation and contraction of an ulcer situated near the biliary papilla. Hæmorrhage may also result from the ulcer, and cicatrisation without stenosis may occur. Of the seventy cases collected by Perry and Shaw, the cause of death was hæmorrhage in nine cases, and perforation in eight cases; the percentage of deaths from hæmorrhage in these cases being much less than that stated by Krause, which was 33 per cent. In the Guy's Hospital cases, stenosis of the gut or bile-duct was found in three cases, cicatrisation of the ulcer was found in eight cases, that is in about 11 per cent. It may be pointed out that the proportion of duodenal ulcers that cicatrise appears to be in great contrast to that of ulcers of the stomach; in post-mortem examinations, according to Brinton, healed ulcers are found to open ulcers in the proportion of about twelve to thirteen.

Symptoms.—Duodenal ulcer may give rise to no characteristic symptoms until it causes death, hæmorrhage, or perforation; and although this may also be said of gastric ulcer, yet the proportion of cases in which an unsuspected duodenal ulcer produces these results is greater than in gastric ulcer. Out of 151 cases collected by Perry and Shaw, in ninety-one there was no record of noticeable symptoms until death occurred by hæmorrhage or perforation. Of sixty cases, there was hæmatemesis or melæna in twenty-three. In many cases there are only vague "dyspeptic" symptoms such as distress after food, and no recognition of the condition is made until melæna or hæmatemesis appear, the patient being considered as suffering from functional disorder of the stomach. In some cases, however, there is great pain following the ingestion of food, pain usually referred to the right hypochondrium outside the stomach region, and associated with local tenderness in that region. It is said that the occurrence of severe pain long after food, that is in about two hours, is suggestive of duodenal ulcer, but this is not a reliable sign. If, however, this kind of pain is associated with melæna, the presence of a duodenal ulcer is rendered more certain. Hæmatemesis may also occur from ulcer of the duodenum, and presents the same characteristic as hæmatemesis from a gastric ulcer, viz. coming on after a meal. Diarrhœa is an occasional symptom in duodenal ulcer; in gastric ulcer, constipation is almost the invariable rule.

Symptoms of Complications.—The perforation of a duodenal ulcer gives rise to the same symptoms as those of gastric ulcer, and may lead to general peritonitis or to a right-sided subphrenic abscess. Stenosis of the duodenum gives rise to practically the same symptoms as those of stenosis of the pylorus; there may be hypertrophy of the stomach, but usually there is great dilatation. The distinction between the two conditions is hardly possible clinically. Stenosis of the bile-duct gives rise to persistent jaundice.

Diagnosis.—The diagnosis of the presence of a duodenal ulcer is not made in the majority of cases; owing either to the absence of distinctive symptoms before a fatal accident occurs, or to the fact that the symptoms of duodenal ulcer are the same as those of ulcer of the stomach. The presence, however, of a duodenal ulcer may be suspected when severe pain follows some time after the ingestion of the food, and there is associated melæna with perhaps hæmatemesis. The association of diarrhœa is not a reliable sign.

Treatment.—The treatment is the same as that of gastric ulcer (*q.v.*). The treatment of hæmorrhage when it occurs is by rest, hæmostatics, and rectal feeding; and the treatment of perforation by operative measures.

CHAPTER XVI.

CANCER OF THE STOMACH (CARCINOMA VENTRICULI).

Definition.—Primary Cancer of the Stomach. Secondary growths also occur in the stomach, but they are rare events. Cancer of the œsophagus only in rare instances spreads through the cardia, and when there are secondary growths in the stomach they are usually an extension through the peritoneum of growths in the neighbourhood of the organ, and commonly appear in the form of multiple nodules. A single large, secondary growth in the stomach is practically unknown.

Etiology.

The etiology of cancer of the stomach is that of cancer generally, and need not be fully discussed in this place. Cancer most frequently attacks those parts of the body which are subject to great changes and to friction; for example, the stomach, the cervix and uterus in the female, the tongue and lips in the male, the intestinal tract at the bends of the colon, more particularly the cæcum and the sigmoid flexure, and the rectum. These conditions, however, can only be considered as predisposing to cancer. Friction of itself will not produce a cancerous growth; there is something else necessary, the exact nature of which is unknown. It has been supposed for example that the development of cancer took place in embryonic cells which had not become differentiated into the adult cells of the organism (Cohnheim). This idea must, however, remain a theory since no proof of it can be forthcoming.

Heredity, however, probably plays some part in the development of a cancerous new growth, but not to a greater extent than a particular locality where the disease prevails. On the other hand, it has been supposed that cancer is an infection, and while the causes mentioned above may be considered as predisposing, the actual development of the growth is the result of an infection of the tissue by a parasite, which has been supposed to be certain oval and encapsuled bodies found in the cancer cell by various observers. Although there is but little doubt that cancer is an infective process, yet it has not been proved that these oval bodies in the cancer cell are the infective agents. The subject cannot be further discussed here, and reference must be made to the numerous original papers on the subject which have been published of late years. As regards the etiology of cancer of the stomach itself but little need be said.

The *age* at which it occurs is that of middle life at which new growths are most frequent in all parts. In 600 cases which were collected by Dr. Brinton, three-fourths occurred between the ages of forty and seventy, and the greatest number between the ages of fifty and sixty. A number of cases, however, have been collected in which cancer of the stomach has been found in young adults. The same remarks are true about cancer of other parts than the stomach.

As regards *sex*, there has been supposed to be a predisposition in males to cancer of the stomach, and this is to some extent correct. In 53 cases collected from the records of University College Hospital, 32 occurred in males, and 21 in females: and a similar proportion in the sexes has been found in other and larger statistics.

Relation to other Diseases.—Previous disease of the stomach has no special relation to the development of cancer. Long continued functional disorder has been considered to predispose to the disease, but there is not the slightest evidence of this. Cancer of the stomach may develop in a chronic "dyspeptic," but no connection can be traced between the two conditions. On the other hand, the association with ulcer and carcinoma of the stomach is of some importance. Although a simple ulcer does not develop after the formation of a cancer in the stomach, yet a certain number of cases of cancer are preceded, perhaps at a long interval, by simple ulcer of the stomach, and at the post-mortem examination it may be found that a scar is present near the new growth, or that the locality of what has previously been the ulcer is occupied by a new growth. Rosenheim considers that 5 per cent of cases of ulcer pass into cancer.[1] There does not appear any advantage in looking at the subject from the point of view of a percentage, and the fact that ulcer of the stomach may be associated with carcinoma being undoubted, the connection between the two must be looked upon simply as the connection between an injury and the subsequent development of a new growth, just indeed as in some cases of carcinoma of the mamma the new growth appears to develop in consequence of a blow upon the breast.

Tuberculosis.—Retrograde tubercle of the lungs is not infrequently found associated with carcinoma not only of the stomach, but of other parts. It may be found in patients who die from other diseases—acute lung diseases, diseases of the circulation, liver, kidneys, etc. The percentage of post-mortems in which retrograde tubercle is found is about 9, according to the statistics of Dr. Kingston Fowler[2] and of myself.[3] By far the larger number of these cases of tubercle are found in patients who die from cancer, and rarely is the tuberculous lesion active; I have met with only two cases of active tuberculosis in cancer, and in both of these the lesions, although recent, were quite insignificant in extent. It has been indeed considered that there is an antagonism between cancer and tuberculosis, but from the results of post-mortem examinations it must be concluded that there is no antagonism to the infection of tuberculosis, but there is an antagonism to its progress in cases where carcinoma is already developed.

Carcinoma of the stomach cannot be considered as related to any other diseases. The acute inflammatory affections which are not uncommonly found in persons dying from malignant disease are secondary to the formation of a tumour. Its relation to cardiac degeneration and atrophy of the kidney are considered below (p. 465).

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[1] *Ztschr. f. klin. Med.*, Berlin, Bd. xvii., p. 116.

[2] "Arrested Pulmonary Tuberculosis," 1892.

[3] *Brit. Med. Journ.*, 31st October 1891.

MORBID ANATOMY.

1. *Nature of the Growths.*—Cancer of the stomach is found in four different forms: scirrhus, cylindroma, medullary carcinoma and colloid carcinoma.

Scirrhus.—Scirrhus forms about three-fourths of the cases of cancer of the stomach. It is a hard growth, often cutting almost like cartilage, and on section shows dense strands of connective tissue, between which are yellowish areas which are the collections of cancer cells. The centre of the growth may be cupped on section, showing that degeneration has occurred, but as a rule the growth is firm throughout. Beginning in the submucous tissue it infiltrates the whole thickness of the stomach, producing not infrequently a firm ring; and it may spread along the wall of the stomach or infiltrate the mucous membrane. It is frequently superficially ulcerated, and the surface of the ulcer may show small fungations with occasionally softened patches. In not a few cases, however, at death, the surface of the growth is not ulcerated, and the appearances which are seen at the pylorus are those of a dense fibroid ring. The diagnosis is made in this case by the examination of a section, when the growth is seen infiltrating the deeper layers of the stomach wall.

Microscopically, scirrhus is characterised by a very dense fibroid stroma enclosing the cancer cells, which are large, nucleated, and oval, pyriform or irregular. Where degeneration has taken place the cells are found granular; they have lost their nuclei and are finally not distinguished as cells, granules only being seen.

Medullary Carcinoma.—Medullary carcinoma originates in the glands of the stomach. It is characterised by large, flat, soft fungating masses projecting from the mucous membrane. It is whiter than scirrhus, and may show points of hæmorrhage in various parts, and most commonly degenerates, a large irregular fungating ulcer being formed on the surface of the growth. Like scirrhus it tends to infiltrate the whole thickness of the stomach wall, but it generally projects into the cavity of the organ. Its general structure is similar to that of scirrhus, but it differs in the cancer cells being more numerous and the stroma being small in amount. It frequently produces secondary growths; but it may, like scirrhus, infiltrate more or less the whole of the extent of the stomach or produce fibroid induration.

3. *Epithelioma* is not a very common growth in the stomach. It forms a soft fungating tumour which ulcerates on the surface like the other growths just discussed. Epithelioma may infiltrate the stomach coats and especially the mucous membrane. It originates in the glands, and microscopically it consists of a slender stroma of connective tissue surrounding a tubular space which is lined by a cylindrical epithelium like that in the glands of the organ. In the early stages the cylindrical epithelium is distinct, but as the growth gets old the regular arrangement of the epithelium is lost and the tubular spaces become filled with cells, the product of the multiplication of the epithelial cells. These undergo various forms of degeneration, and may form small cysts containing granular material and liquid.

4. *Colloid Cancer.*—Colloid cancer is present in about 9 per cent of the cases, although Brinton found 17 cases out of 100 cases of cancer of the stomach. It is seen either as a tumour projecting from the mucous membrane or as a diffuse infiltration of the stomach wall. Its appearance is very characteristic, inasmuch as the stroma of the tumour surrounds transparent, gelatinous looking masses which consist of the cancer walls in a condition of colloid degeneration. The connective tissue stroma likewise undergoes colloid degeneration. Colloid cancer is the form in which disease occurs in young adults, the other forms occurring in middle and old age.

II. *Seat of the New Growth.*—From a clinical point of view the seat of the new growth is more important than its nature, since all the forms of carcinoma of the stomach lead to death; and, from the position of the growth, the effect on the stomach and consequently the signs and symptoms vary. The parts of the stomach where the new growth may be situated are in the order of frequency, the pylorus, the lesser curvature, the cardia, and the greater curvature. But there is another form of cancer which is diffuse, infiltrating the stomach generally, and this may occur from any of the forms described above, although it is most frequent with scirrhus and medullary carcinoma.

The frequency with which the pylorus is affected is variously given in the statistical records. From an examination of 360 cases Brinton found that the pylorus was affected in 60 per cent; while Hahn found it affected in only 35 per cent. It may be concluded, however, that in the majority of cases of cancer of the stomach the pyloric portion is affected more frequently than the other parts. According to Hahn the lesser curvature is affected in 16 per cent of cases, and according to Brinton, the cardia is affected in 10 per cent, while the greater curvature is but rarely affected by the disease.

III. *Changes in the Growth.*—The chief change that occurs in the growth in the stomach is one of degeneration and of ulceration, and this is not unimportant, inasmuch as the growth which has previously produced stenosis of the pylorus may ulcerate so as to again open the passage for the stomach contents.

Another change which occurs in the growth is the formation of adhesions to the neighbouring parts, and the spread of the growth along the adhesions to the neighbouring organs and lymphatic glands. The most important adhesions occur between the liver and the stomach, and the stomach and the tranverse colon. But in some cases all the parts round the pylorus may be matted together in a firm mass in which the omentum is involved; this occurs not infrequently in cases of scirrhus. The importance of these adhesions is that it renders the tumour of the stomach immovable, and thus alters its clinical signs, and that along them the growth spreads and involves the liver and the neighbouring parts.

Adhesion to the colon not unfrequently results in a fistula being formed between the stomach and the colon; adhesion to the abdominal wall may occur and an external fistula may be formed, while in ulceration of the growth perforation of the stomach may occur and subphrenic abcess result.

The other effects of the local extension of the growth are chiefly thrombosis of the inferior vena cava resulting from pressure, and pressure and obstruction of the thoracic duct. The portal vein or one of its main branches may also be obstructed.

Secondary Growths.—In carcinoma of the stomach secondary growths most commonly occur in the parts immediately around the tumour. Altogether they occur in about 48 per cent of the cases. They are most frequent in the *liver* owing to its proximity to the tumour and to the frequent adhesions which are formed between the stomach and the liver; growths may, however, occur in the liver without adhesions being formed. In 53 cases collected from the records of University College Hospital, the liver was affected in 18 cases, or 30 per cent. The *peritoneum* not infrequently shows cancerous nodules; *e.g.*, in 17 per cent of the cases just referred to. In some cases the cancerous peritonitis may be limited at death to the pelvic region; in other cases it is diffused over the whole of the peritoneal cavity. It may sometimes be shown in great thickening of the peritoneum in the upper part of the abdomen, and it may lead to constriction of the stomach.

The *lungs* frequently show growths in the form of small discrete nodules, which may be dry and firm as in cases of epithelioma or larger and soft as in cases of medullary carcinoma. Sometimes they project from the surface of the lung, as in the liver, in the form of umbilicated nodules.

Other Effects of the Growth.—*Ascites* may be present in the later stage of cancer of the stomach. It is due chiefly to cancerous peritonitis, or to obstruction of the portal vein by the pressure of enlarged glands or of a new growth in the liver. *Jaundice* is present in some cases where the liver is affected, and may be due to pressure on the bile-duct or to large growths in the liver substance. Not unfrequently in post-mortem examinations of cancer of the stomach, as in other cases of cancer, the kidneys are found to be in a state of cirrhosis; being small, granular on the surface with an adherent capsule and a diminished and tough cortex. The heart muscle is frequently in a state of fatty degeneration. Both these conditions are found, however, in the post-mortem examinations of old people dying from various causes, and the fatty heart may be found in simple abdominal tumours, when they are large and have existed for a long time.

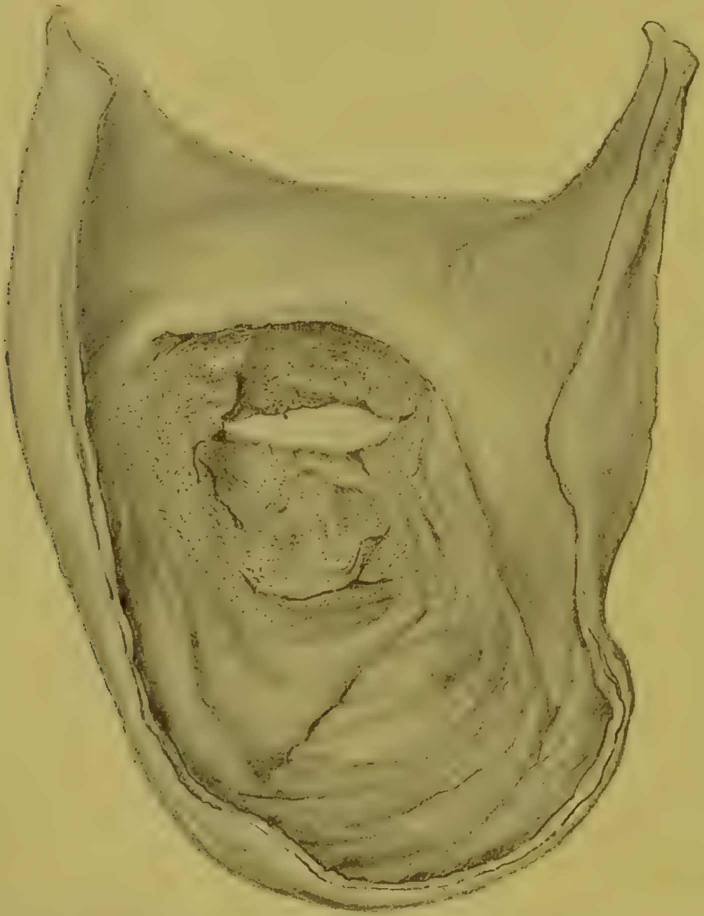

Fig. 51.—Cancer of the pylorus. (From a preparation in the Museum, University College.) About natural size. The growth surrounds the orifice and is pitted on the surface: the mucous membrane round the growth is superficially ulcerated. The antrum pylori is dilated.

Effects of the Growth on the Stomach.—The results of the formation of a cancerous new growth in the stomach may have several effects on the organ itself, which may be classified as (1) the effects of the growth itself, (2) the effects of the growth on the functions of the stomach.

1. *Effects of the Growth itself.*—These vary according to its

size, its position, and the mode in which it infiltrates the walls of the organ.

Growth causing Obstruction of the Pylorus.—The commonest cause of obstruction of the pylorus in cancer is the development of scirrhus around the orifice (Fig. 51). In the great majority of cases the pylorus is found obstructed at death. The growth does not extend into the duodenum, but by infiltrating the surface narrows the orifice or produces a narrow tract, sometimes not as large as the little finger. Obstruction of the pylorus also occurs in fungating growths near the orifice, which act as a mechanical hindrance to the passage of the stomach contents into the duodenum. The obstruction in this case is much less than when a stricture is formed. The result on the stomach of obstruction to the pylorus is dilatation and hypertrophy, but the extent to which dilatation and hypertrophy may individually occur varies (see Dilatation of the Stomach, Chapter XIV. p. 380). In the majority of cases there is some dilatation during life, and after death dilatation is frequently the the chief condition found. Hypertrophy may, however, take place to so great an extent that the stomach remains small,

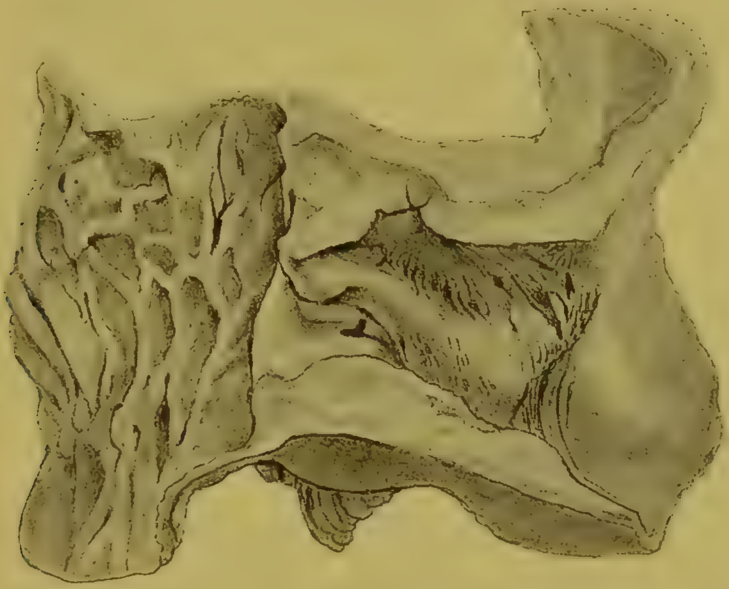

FIG. 52.—Cancer of the pylorus. (From a preparation in the Museum of the Royal College of Surgeons. By permission of the Council.) The stomach wall for several inches from the pylorus is greatly thickened. The thickening is due partly to hypertrophy of the muscle, but chiefly to the cancerous growth, which is irregular and ulcerated on the surface. The pylorus is greatly constricted. ⅗ natural size.

and hypertrophy is frequently seen limited to the pyloric region, and not extending to the cardiac (Fig. 52). When hypertrophy does not occur, and dilatation alone is present, it may be enormous, so that the stomach fills two-thirds of the abdominal cavity. In some cases the tumour drags the stomach out of position, so that the lesser curve is nearly at the level of the umbilicus. This occurs in tumours of the pylorus, and only when they are non-adherent.

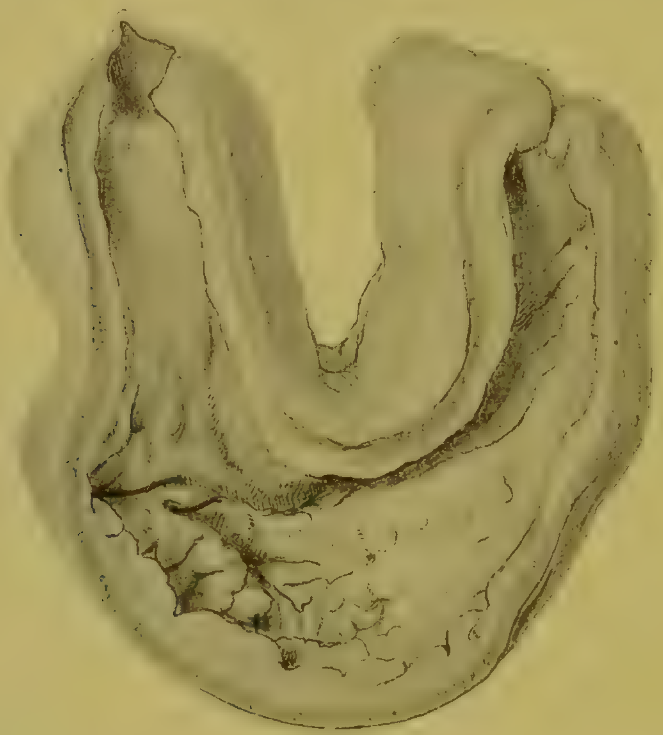

FIG. 53.—Diffuse cancer of the stomach. (From a preparation in the Museum, University College.) About ⅔ natural size. The stomach is the shape of a leathern bottle; the cardia being on the right and the pylorus on the left of the figure. Both the œsophagus and duodenum are normal. The stomach walls are greatly thickened throughout, and as in the specimen shown in Fig. 54, the thickening is due to an increase of the mucous and submucous coats, with great hypertrophy of the muscular coat. Microscopically, the appearances are very similar to those shown in Fig. 55, cancer alveoli being seen in the submucosa chiefly. The hypertrophy of the muscularis is extreme, and there is a large increase of fibrous tissue throughout the organ (scirrhus).

In some cases with slight pyloric obstruction diffuse infiltration of the stomach wall along the mucous coat occurs so that the stomach assumes the form of an oval bag in which its shape is lost (Fig. 53). These cases are always associated with fibroid induration of the walls and with hypertrophy, and it is noticeable that these latter changes extend only so far as the cancer itself has infiltrated. This is well shown in Fig. 54, in which two-thirds of the stomach from the pylorus are thickened and cancerous, while the cardiac end is thin and normal.

Growths in the Mid-region of the Stomach.—When the cancer is situated on the lesser curve, or on the posterior surface (Fig. 56), its effect on the stomach is less than when there is pyloric stricture. Dilatation may occur in these cases, but its causation is the same as that described in simple dilatation, viz. either from the effect of the nervous system, or by a muscular weakness produced by the presence of an infiltrating new growth.

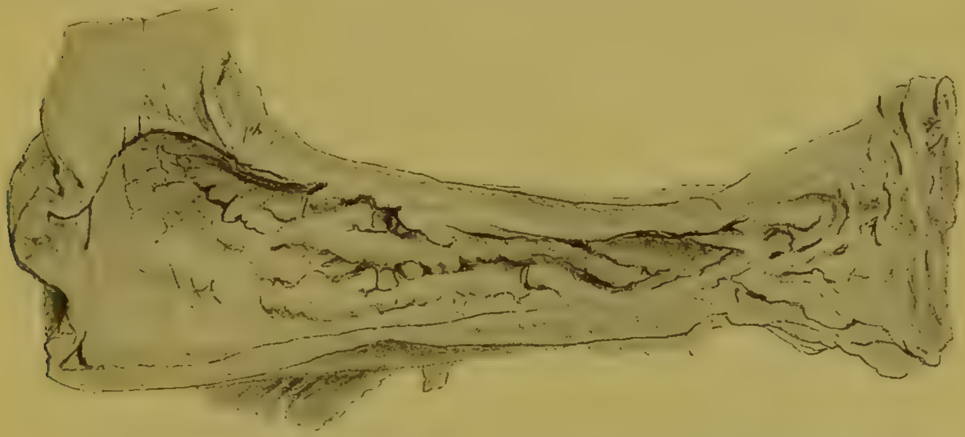

FIG. 54.—Diffuse cancer of the stomach. (From a preparation in the Museum, University College.) About ½ natural size. The wall of the organ is thickened from the pylorus to the cardiac pouch, the thinness of which is in great contrast to the thickness of the greater part of the organ. The greatest thickening is at the pylorus, and is seen to be due not only to an increased size of the mucous and submucous coats, but to great hypertrophy of the muscular coat. The surface of the mucous membrane shows irregular rugæ. Microscopically, the thickening of the wall of the stomach is seen to be due to scirrhus infiltrating the mucosa and submucosa (Fig. 57).

Obstruction of the Cardia (Fig. 57).—Obstruction of the cardiac orifice occurs in new growths situated round it, exactly as in stricture of the pylorus. The stomach is in this case small, contracted, and not hypertrophied, while the œsophagus above the stricture is dilated and hypertrophied.

Perforation.—Perforation of the stomach in cancer occurs in about 4 per cent of the cases, according to Brinton; it may result in a subphrenic abscess or in a gastro-colic fistula, or, more rarely, in a gastro-cutaneous fistula.

II. *The Effects of the Growth on the Functions of the Stomach.*—Weakness of the muscular coat of the organ is a frequent sign in cancer of the stomach, and is due partly to the presence of the solid mass in the stomach wall, and partly to diffuse fibroid and carcinomatous infiltration of the stomach wall. It is present in dilatation when this occurs in pyloric stenosis; and it is also caused by the general condition of the patient, the anæmia, weakness, and cachexia produced by disease.

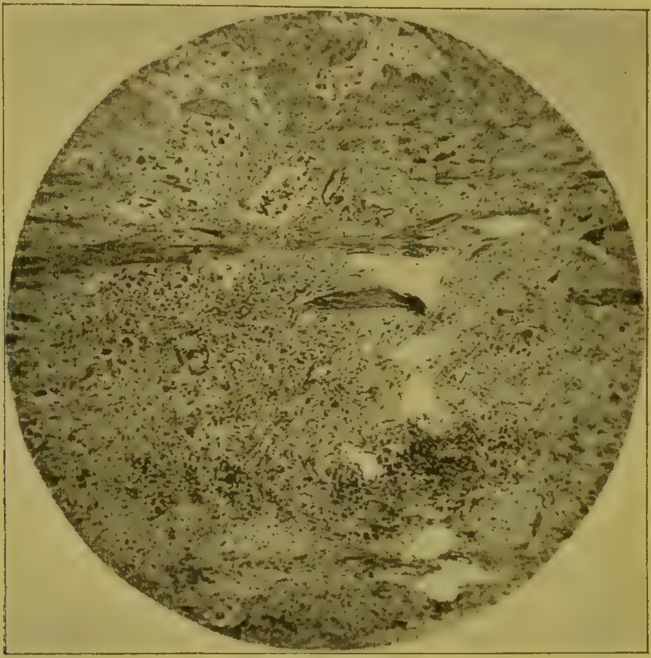

FIG. 55.—Diffuse carcinoma of the stomach, from the preparation represented in Fig. 54. From a photograph, × 40. The figure shows the junction of the mucosa (above) and the submucosa (below); the separation between the two being marked by bundles of fibres of the muscularis mucosæ which is broken up by the infiltrating growth. The submucosa is greatly thickened and shows alveoli filled with cancer cells; these are also seen in the depth of the mucosa. From a preparation hardened in alcohol and stained with logwood.

Secretion is affected in various ways. The secretion of hydrochloric acid may not at once be affected, and the digestion may at first be practically normal; but as the disease progresses, sometimes early in the case, the hydrochloric acid diminishes and may disappear. The absence of free hydrochloric acid from the stomach contents may be partly due to the alkaline discharges of the ulcerated tumour neutralising them, as suggested by Maly; but it is also due to the infiltration of the mucous membrane by new growth, although in not a few cases of cancer of the pylorus there is no evident affection of the cardiac region where hydrochloric acid is secreted. It is probable that the diminution of hydrochloric acid which is observed in many cases of cancer is due to the effect of the general condition of the body on the secretion in the stomach,

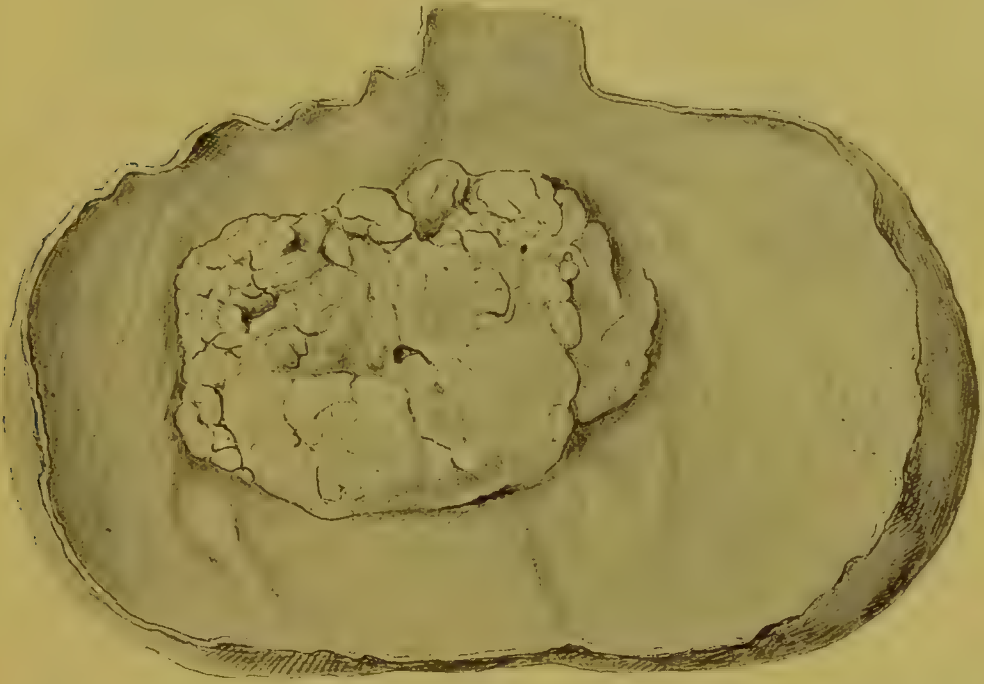

Fig. 56.—Cancer of the posterior wall of the stomach. (From a specimen in University College Museum.) About ⅔ natural size. The growth forms a fungating mass slightly ulcerated on the surface, and situated nearer the lesser curvature than the greater. The stomach is not dilated and there is no hypertrophy. There were very few symptoms referable to the stomach during life.

and also to the association of atrophy of the glands, of dilatation, and of catarrh with the disease.

Although, therefore, in some cases the digestion may be normal, in most there is great deficiency in the motor power of the organ, and a deficiency of hydrochloric acid. The deficiency in motor power and in secretion leads to delay of food in the organ and bacterial fermentation of food. This is most marked in dilatation, especially in that extreme form produced by stenosis of the pylorus, and in such cases the acid products of bacterial fermentation, chiefly lactic acid, are found, and their presence in the stomach contents tends to hinder the secretion of hydrochloric acid (Chapters. III. and IV.). Similar changes also occur in diffuse cancer, in which the organ is contracted.

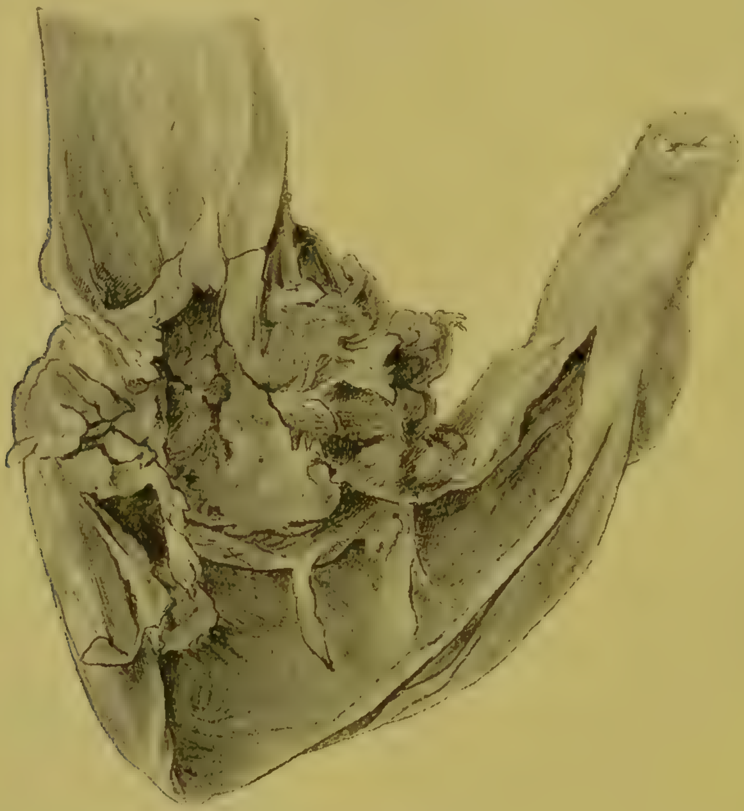

FIG. 57.—Cancer of the cardia. (From a preparation in the Museum of the Royal College of Surgeons. By permission of the Council.) $\frac{2}{3}$ natural size. A ragged growth surrounds the cardia, ulcerated on the surface. The stomach wall is infiltrated, and the growth has invaded the peritoneum. The pyloric region is contracted, the œsophagus is dilated and hypertrophied.

SYMPTOMS.

In the description of the symptoms caused by ulcer of the stomach it was seen that some were produced by the actual presence of the ulcer in the organ, viz. pain after food, tenderness, and hæmorrhage. And in cancer of the stomach there are also special symptoms which are due to the presence of the malignant disease, which do not differ from those observed in similar forms of malignant disease occurring in other parts of the body.

The signs and symptoms of malignant disease may be described under three headings :—(1) Those due to the growth itself; (2) those due to the effect of the growth on the body, either as interfering with the functions of an important organ or as toxic in their origin; (3) those due to complications which arise either in connection with the growth itself or secondary to its effects.

In all cases the onset of the symptoms of malignant disease is insidious, so that in the majority of cases there is a period of slight symptoms of indefinite nature followed by a period of pronounced symptoms, due to the spread of the growth and its effects; during this second stage both the nature and the locality of the growth may be diagnosed. This stage is again followed by a third stage preceding death, in which complications frequently arise, and these complications may to a greater or less extent mask the symptoms and physical signs of the second stage of the disease. It is of great importance to bear in mind this clinical course of malignant disease, inasmuch as the patient may be seen for the first time in any one of the three stages. In the first and third stages a diagnosis is frequently impossible; in the first stage because of the indefiniteness of the symptoms, and in the third stage because the symptoms are masked by those of complications. For example, malignant disease of the colon may be masked by the signs and symptoms of a perforative peritonitis; cancer of the stomach may be masked by ascites, by perforative peritonitis, by acute lung affections and by large secondary growths in the liver or by other conditions which will be fully discussed afterwards. It is in the second stage of the malignant disease that the symptoms and signs are characteristic and that the recognition of the disease is usually made.

(1) *Signs and Symptoms due to the Growth itself.*—These are pain and tenderness, hæmorrhage, and the existence of a primary tumour and of secondary tumours.

Pain.—The pain of a malignant growth is usually sharp and shooting. Sometimes when it infiltrates solid tissues—for example, bone or a solid organ, the pain may be dull and boring in character, or it may be in some cases confined to a sense of weight and oppression as in malignant disease in the thorax. The pain is *local*, and even when diffuse its point of greatest intensity is over the seat of the disease; it is accompanied by local tenderness which may be great or slight.

Hæmorrhage.—The malignant tumours differ from all solid benign tumours in the fact that they lead to frequent hæmorrhages. These are due sometimes to the rupture of small vessels in the tumour itself, and in this case there are frequent and small hæmorrhages, as in ulceration of the growth; and in some cases to the invasion of a large vein or artery which becomes ruptured, and leads to profuse and sudden hæmorrhage, ending usually in death.

The Existence of a Tumour.—The characteristics of the tumour in malignant disease are that it is irregular, hard, and tender. Although it may at first be movable, when, for example, it occurs in the abdomen, it subsequently becomes fixed owing to the infiltration of the growth into the surrounding parts. Manipulation not only causes pain, but may produce hæmorrhage. The tumour enlarges; and in an irregular manner, forming nodules which can be frequently felt extending from the main mass of growth. Secondary tumours are also one of the characteristics of malignant disease, they are found not only in the lymphatic glands, but also in many of the solid organs, and they may form the first diagnostic sign of malignant disease.

The special signs and symptoms which malignant disease of a particular organ produces are due to the effect of the growth on the functions of the organ, and, if this is a hollow viscus, on the passage of its contents.

(2) *Signs and Symptoms due to the Effect of the Growth.*—It is always very difficult to estimate how far the symptoms of malignant disease are due to the toxic effect of the growth itself, or simply to an interference with the functions of an important or a vital organ. Probably the symptoms are due to both these causes, but no one who has had any experience in the post-mortem examination of persons dying of cancer who have been the subject of operation or not, can doubt that in many cases the extent of cancerous disease does not account for the fatal result. Although many of these cases are associated with degeneration of the kidney (cirrhosis), or with fatty degeneration of the heart, yet it appears that some effect

must be ascribed to the growth itself. On the other hand, there can be no doubt that the more vital the organ affected, the more severe are the symptoms and the more rapid the death.

Uncomplicated cancerous disease does not of itself produce fever. Fever is observed in the course of some cases, but it is usually due to complications which have arisen; localised abscesses near the new growth, peritonitis, or some inflammatory condition of the lungs. In those cases, which have been the subject of fever during life, one or other of these conditions is usually found after death. The remaining symptoms due to the effect of the growth on the body are classed under the heading of *cachexia*, the main features of which are wasting, muscular weakness, and anæmia. Anæmia varies with the extent and increase of the malignant disease, and in its advanced stage the peculiar waxy pallor of the skin in general is produced, with sometimes an earthy tint; a waxy pallor which in many cases is not unlike that observed in pernicious anæmia. Associated with it there is an anxious expression of the face due to the pain from which the patient suffers. The weakness, slight at first, gradually increases, and is associated with myotatic irritability. The wasting is with the anæmia and weakness characteristic of malignant disease; and it is general, the fat disappearing and the muscles becoming thin and flabby. Not unfrequently, however, a fair quantity of subcutaneous fat is present at the time of death, even when there has been great wasting of the muscles. The other signs and symptoms of the effect of the growth are special, that is, are due to the effect of the growth on the organ invaded.

(3) *Signs and Symptoms of the Complications.*—Malignant growths when they affect a hollow viscus, such as a part of the alimentary tract, are specially liable to be invaded by micro-organisms; this occurs more particularly when the growth ulcerates. Invasion takes place through the base of the cancerous ulcer, which may be very thin when it affects a hollow viscus; or it may take place through an acute ulcer formed at the side of the cancerous growth, or even some distance from it, when this causes obstruction of the passage. This acute ulcer is due to bacterial infection and the reten-

tion of the contents of the gut. The perforation of such an ulcer may occur. According as one or other of these events happens there may be a localised abscess formed near the cancerous growth, or there may be a diffuse inflammation, or a localised gangrene. Instances of these may be cited in the case of malignant disease of the uterus causing pyosalpinx; malignant disease of the stomach causing localised abscess near the growth; and malignant disease of the larynx or œsophagus causing localised gangrene. Other acute affections are frequently observed in malignant disease; these are both pulmonary and intestinal. Pneumonia and bronchitis are not infrequently the immediate cause of death, and acute diarrhœa due to bacterial decomposition or infection in the intestines may also be the immediate cause of death, not only in cases of cancer affecting the alimentary tract, but in cancer of any part.

(4) *Course and Duration of Cancerous Disease.*—The course of malignant disease is progressively downwards. There are intervals, however, of remission of the symptoms brought about either by operation, by the improvement of the patient's general condition, or by remedying the functional defects produced by the growth. The intervals of relief which are afforded by operation vary in length and need not be discussed here, but they are chiefly observed in cases of external cancer.

The duration of cancerous disease varies somewhat; when it affects an internal organ and a vital one, such as the stomach or the liver, the average duration may be put down at eighteen months or two years.

Causes of Death.—Patients who are the subjects of malignant disease, in the majority of cases, show at the post-mortem examination large primary or secondary growths. Death may also be caused by local accidents in the primary or secondary growths, for example by hæmorrhage, by bacterial infection producing abscesses or causing gangrene, or by the pressure of the growth on important vessels. In not a few cases, however, death occurs from remote complications, either from those mentioned above—pneumonia and bronchitis—or from the results of fatty degeneration of the heart as shown in cardiac failure, or in uræmia from granular contracted kidney. In some cases, therefore, at the post-mortem examination the size of the growth may be insignificant when these diseases of the heart and kidney are found, and this is especially so in the case of malignant disease of the breast, tongue, or cervix uteri.

Symptoms of Malignant Disease as it affects the Stomach.—These show an infinite variation. The chief point to be borne in mind is that they consist in the association of gastric symptoms with those of malignant disease which have just been considered. The symptoms of cancer of the stomach may be at first slight and limited to indefinite "dyspeptic" symptoms, but although these slight symptoms may not in some cases alter greatly in character during the course of the disease, yet associated with them are the progressive symptoms of malignant disease. In the majority of cases, however, the early dyspeptic symptoms pass into symptoms of a more definite nature which are associated with the effect of the new growth on the functions and structure of the stomach. These typical symptoms are local pain, vomiting, and hæmatemesis. A typical case of cancer of the stomach may be described as follows:—The patient, male or female, middle-aged, and not uncommonly with a history of previous good health, commences to suffer from symptoms of indigestion of food, the chief signs of which are loss of appetite, which may be extreme, some nausea, and pain in the chest after eating. For some time the symptoms remain in this stage, perhaps not improving or getting worse, but the effect of the symptoms is out of proportion to their severity; that is, the patient gets weaker, wastes, and gets paler, while there is no physical sign of organic disease in the thorax or abdomen, and no renal disease to account for the effect on the general nutrition. Vomiting may now occur, irregular in its onset, and not definitely associated with the ingestion of food; the vomit containing an excess of organic acids, chiefly of lactic acid. On inflating the stomach with gas it is found that it is dilated, passing below the umbilicus, and into the axillary region. Epigastric pain is now not infrequently emphasised, not aggravated in any way by the taking of food, but continuous and of a lancinating character. Pain from this time onwards is continuous, being frequently exaggerated in paroxysms. At this time, or it may be later or earlier, a tumour of the epigastrium is discovered over the pyloric region, or over the mid-region of the stomach; at first it is suspected only by the presence of an area of increased resistance—later on it is shown by the presence of nodules, or of a large nodular mass. The patient is now in, what has been called above, the second stage of malignant disease, that is, a stage of the fully developed disease in which in addition to the symptoms that have been mentioned referable to the stomach the weakness and the waxy anæmia of malignant disease are present. From the middle stage onwards the condition of the patient varies. Hæmorrhage from the stomach is a varying symptom, and usually the chief signs are those due to the progressive growth of the tumour, and the dilatation of the organ associated with bacterial fermentation of the food. The patient gradually sinks, becoming weaker and weaker, and death occurs from exhaustion, from hæmorrhage, or from the incidence of an inflammatory disease.

It is necessary now to discuss the symptoms of cancer of the stomach in more detail.

Anorexia.—Loss of appetite is an important symptom in cancer of the stomach, inasmuch as it constitutes one of the distinguishing features from ulcer. In ulcer the appetite is not lost, it may even be increased, and indeed, although patients say that they cannot eat, this is due to the fact that they are afraid to eat owing to the pain produced. According to Brinton, loss of appetite is present in 85 per cent of the cases of cancer of the stomach. It is a variable symptom as regards its onset; it usually appears early in the case, but it may be late, and in the last stage of the disease it is usually well marked. The loss of appetite is no doubt greatly affected by the treatment of the patient.

Tongue.—The tongue presents a varied appearance, it is sometimes thickly coated, but is usually broad, pale, flabby, tooth-indented, and covered with a light fur.

Pain.—Pain in cancer of the stomach is present in 92 per cent of the cases (Brinton). It is lancinating in character; but this varies somewhat, as it may be dull and gnawing, or may consist simply of a sense of weight and oppression and tightness. Its seat is in the epigastrium and is both local and diffuse; it is most intense over the growth itself and spreads over the stomach region. Like the pain of ulcer it may spread through to the back and be felt in the lower dorsal region on the left of the spine; this is said to be especially the case in cancer affecting the lesser curve and the posterior surface of the organ (Brinton). The pain is associated with local tenderness, the degree of which varies greatly in different cases. As a rule it is indefinite, the local tenderness that is elicited on the manipulation of the tumour being most commonly found over the pyloric region; but in some cases there is a more defined local tenderness, and this is associated, according to Brinton, with ulceration of the growth. This sign, however, cannot be relied on for the diagnosis of ulceration of the growth. The pain differs from that of ulcer (1) by the fact that it is not related to the ingestion of food; (2) it is more or less continuous and but rarely shows the defined localisation of the seat of pain and of tenderness as in cases of ulcer; and (3) it is not to so great an extent relieved by vomiting.

Vomiting.—Vomiting occurs in 87 per cent of the cases. Its causes are, first, the presence of the growth in the stomach wall, and, secondly and chiefly, the presence of dilatation of the organ. It is also present in contraction of the stomach due to cancerous cirrhosis. Vomiting is chiefly a symptom of malignant disease of the pylorus in which there is dilatation of the organ and great delay of food in the stomach with bacterial fermentation. The chief characteristic of vomiting in cancer is that it is irregular, bearing no direct relation to the ingestion of food, especially when there is great dilatation. Moreover, it gives no relief to the pain, and in this point differs greatly from the vomiting of gastric catarrh and of ulcer. The vomited matters almost invariably show the products of bacterial fermentation, that is, chemically they are very acid and contain no free hydrochloric acid, but a large amount of organic acids, lactic, acetic or butyric; they contain also peptones and undigested food, and microscopically show large numbers of bacteria and sarcina and cells in various stages of degeneration. It is impossible to diagnose the presence of cancer cells in the vomit, and a fragment of the tumour is but rarely brought up in a condition which is suitable for a satisfactory microscopical examination. Pieces of the tumour have been brought up by the sound in cases of cancer of the cardia, but even this is a very rare event. (For further details regarding the vomited matters in the cancer, see Chapter IV. pp. 93 and 97.)

Hæmatemesis.—Hæmatemesis occurs in 35 to 40 per cent of the cases, and is thus much less frequently observed than in ulcer.[1] The amount of blood lost, however, varies. As it usually comes from the surface of the ulcerating growth, the bleeding is slight, and the blood oozes from the growth and generally collects in the stomach, being changed before being vomited. Vomiting may not occur, however, for some time after the hæmorrhage, so that a large amount of "coffee-grounds" vomit may be brought up, which does not make the condition of the patient any worse at the time, as it is the accumulation of small hæmorrhages which have been going on for some days. In other cases, as when a large vessel is opened by the growth, profuse hæmatemesis of bright red blood or of clots of blood takes place, and this may be fatal. Hæmatemesis, as in the form of "coffee-grounds" vomiting, is not infrequently associated with dilatation of the stomach, so that in the vomited matters there are the organic acids and bacteria previously described. The hæmatemesis of cancer differs from that of ulcer in some points. What may be described as the characteristic hæmatemesis of ulcer is the bringing up of moderately large quantities of red or clotted blood associated with melæna, the occurrence of the hæmorrhage being associated with the taking of a meal or one of the other conditions previously described (Chapter XV. p. 428). In cancer the hæmorrhage has no particular association with a meal, although it may be induced by manipulation of the growth.

Wasting.—During the earlier stages of malignant disease of the stomach there is progressive wasting. Afterwards the patient may improve by treatment and may actually gain weight, but he never recovers his original weight, and the gain of weight is very slight and is soon lost again. In other cases the emaciation is rapid and progressive. The gain of weight during treatment may be as much as six or seven pounds, and is due to the fact that the functional disorder of the stomach is remedied, and the patient can assimilate more food than he has previously done; this is accomplished by proper dieting, by rectal feeding, or by the treatment of the dilated stomach and of bacterial fermentation. The total loss of weight during the whole course of disease of the stomach varies. No exact figures can be given, and some subcutaneous fat may still be present when the patient dies or it may be quite absent, but the total loss of weight during the course of the illness may be as much as forty-two pounds.

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[1] In the 53 cases already quoted (p. 465), hæmatemesis occurred in 20, or about 36 per cent. Brinton gives the percentage of hæmatemesis in cancer as 42.

The general symptoms other than wasting have already been discussed under the heading of the symptoms of malignant disease generally (p. 473). There remains now only to consider the condition of the bowels and the urine.

The *bowels* are usually constipated. Diarrhœa occurs in about 35 per cent of the cases. It has been ascribed to the acrid discharges from the cancerous tumour irritating the intestine, but it has at least two other causes, viz. the expulsion of the foul contents of the stomach in bacterial fermentation into the small intestine, and the setting up of bacterial decomposition of its contents. It may alternate with constipation as in simple functional disorder of the stomach. Melæna is not a common symptom, but may be present when there is profuse hæmorrhage.

The *urine* shows no great change from the normal except in cases of dilatation and bacterial fermentation, in which there is an increase in the ethereal hydrogen sulphates, an increase in the amount of indigo-forming substances, and frequently also albumosuria (Chapter IV. p. 119).

Physical Examination.—The methods of physical examination are the same as those employed for ulcer, viz. inspection, palpation, percussion, and auscultation. But two other methods are of great value in the investigation of the stomach in cases of cancer, viz. inflation of the stomach in cases of dilatation (Chapter XIV. p. 387), and the use of a test-meal to determine the activity of the digestive process (Chapter V. p. 149).

Inflation of the stomach is of use in determining the degree of dilatation of the organ in doubtful cases; it is also of great service in the diagnosis as to whether the tumour is present in the stomach or outside the organ.

The test-meal is used to decide the question of the delay of food in the organ, to determine the amount of hydrochloric acid secreted during digestion, and the presence of bacterial fermentation and its products. The examination of the vomited matters frequently gives as much information as the use of the test-meal.

Physical Examination of the Abdomen.—Two classes of cases are met with: (1) cases of cancer where a palpable tumour is absent, but there is dilatation of the organ; (2) cases of cancer where a tumour is present, with or without dilatation of the organ.

(1) *Cases without Palpable Tumour and with Dilatation.*—The different methods of physical examination in this case only reveal the signs of dilatation of the organ which have been already fully discussed (Chapter XIV.), so that they need not be further referred to. But it may be said that inspection in cases where the pylorus is obstructed shows great prominence of the abdomen not uncommonly in the umbilical and epigastric regions, and palpation may show no more definite sign than the splashing which is present in dilated stomach with perhaps some increased resistance and some tenderness over the pyloric region.

(2) *Cases of Cancer with Tumour, with or without Dilatation;* (a) *Without Dilatation.*—The stomach itself may form a tumour. This occurs in diffuse infiltration with cancer, associated as it is with fibroid infiltration and with hypertrophy of the muscular coat. The physical signs observed are the following:—

Inspection shows no enlargement of the abdomen, no dilated veins on the surface. *Palpation* reveals an oblong, firm, and uniform mass stretching from the left hypochondrium across the epigastrium towards the umbilicus; or, in other cases, an almost transverse oblong mass, the lower edge of which is firm and rounded, the upper edge being ill-defined. The tumour is frequently tender. The size and distinctness of the tumour may vary from time to time, according to the amount of food in the organ.

Inflation of the stomach produces no dilatation of the organ, and may cause great epigastric pain and distress.

Percussion.—The note over the tumour is usually dull but on deep percussion a tympanitic resonance may be elicited.

Auscultation.—On listening over the stomach when the patient drinks water the fluid may be heard to enter the organ with a splash as in a dilated stomach.

Lastly, *the test-meal* shows diminished digestion of food, the presence of a diminished quantity of hydrochloric acid, and an excess of the acids of bacterial fermentation, lactic acid especially. A similar tumour is formed by a thickened omentum and by non-cancerous cases of cirrhosis ventriculi (Chapter XIV.). From thickened omentum, the stomach tumour is distinguished by the results of inflation and by the splash heard on auscultation (p. 491).

(b) *Localised Tumours of the Stomach, and Tumours with Dilatation of the Organ.*—Localised stomach tumours may be discovered in various parts of the organ, commonly at the pylorus, but also along the lesser curve and in the cardiac region. Most of these tumours, especially those at the pylorus, are associated with dilatation of the organ; but as the physical signs of dilatation have already been fully discussed (Chapter XIV.) attention will now be confined to the solid tumours affecting only part of the organ. Such tumours have certain common characteristics. The size varies, but they are hard and irregular; they are movable, tender, and sometimes move on deep respiration. They vary—especially tumours of the pylorus—from day to day as regards their position and the ease with which they may be felt, and the effect of inflation of the organ is frequently to obscure the physical signs of the tumour.

Tumours of the Pylorus.—Inspection gives no evidence of the pyloric tumour. By palpation the mass is felt in the pyloric region towards the right hypochondrium, above and to the right of the umbilicus. This is the usual position of a pyloric tumour; but it may be under the liver within the costal margin, and be discovered only as a fulness in that region, or it may be lower down in the abdomen, being situated near the umbilicus or even below it. The mobility of the tumour varies; in some cases it is fixed in one position, but in not a few cases it is movable with the hand and alters with the position of the patient, and may exhibit extreme movability, over a radius perhaps of 4 or 5 inches. These very movable tumours are always situated at the pylorus, and there are no other tumours in the upper part of the abdomen which exhibit this physical sign, with the exception of movable kidney. Not infrequently the tumour moves with respiration, descending on deep inspiration, but not being affected much by expiration. If, however, the tumour is attached to the liver it moves downwards in inspiration and upwards in expiration. It is hard and tender, and varies in size from a mere nodule to a mass the size of a fist or larger.

A valuable sign in tumours of the pylorus is the gurgling of gas through the tumour, which may be brought about by manipulation of the stomach; this is frequently observed even before inflation of the stomach. The results of inflation on the pyloric tumour is to obscure its physical signs or altogether to obliterate them. Sometimes they are pulsatile, a quality which is given to them by the proximity of the aorta.

2. *Tumours of the lesser curve are discovered* by palpation, and are felt as a nodular mass, varying in extent, and situated across the abdomen just above the umbilicus, when the stomach is dilated and descends in the abdomen. The tumour descends on deep inspiration, but does not move upon expiration. As regards hardness and tenderness, it presents the same characters as the pyloric tumour, and inflation of the stomach demonstrates the position of the tumour with regard to the stomach which lies below it.

Tumours of the Cardiac Region.—These are of two kinds: as to whether they are round the cardiac orifice causing stenosis or diffused over the cardiac pouch. In the first case the stomach is small, the abdomen is not distended, and there is no tenderness over the epigastric region. The passage of the sound shows obstruction to entering the stomach, and particles of the tumour may be removed by the sound which, microscopically examined, will clear up the case. The physical signs generally are those of cancer of the lower part of the œsophagus; regurgitation of food occurring, not infrequently containing blood, especially after the passage of the sound. The regurgitated liquid consists of undigested food, and contains no free hydrochloric acid and no peptone (see Chapter V.). In the second case where the tumour is diffused over the cardiac pouch, it is felt as an irregular nodular mass in the left hypochondrium, its position being more accurately defined in relation to the stomach by inflation of the organ. Palpation and percussion will show the tumour passing under the margin of the left ribs and thus simulating a splenic tumour. This, however, is a rare condition, and in many cases tumours of the cardiac region cannot be discovered by external physical signs.

The tumours which have just been discussed are discovered as small nodules or as larger nodular masses. The size of the tumour discovered by palpation is no criterion of its actual size; as after death such tumours are always found to be larger than physical examination of the abdomen has shown. In not a few cases there is no distinct nodular tumour, but a great increase of resistance over one part of the stomach, especially near the pylorus, which from the signs cannot be diagnosed as a new growth. In other cases, especially in growths on the posterior wall, no tumour is felt during life (Fig. 56).

SYMPTOMS OF COMPLICATIONS.

1. *Secondary growths* occur in the liver, the peritoneum, and the lungs. They also occur in the retro-peritoneal lymphatic glands, but the enlargement of these glands, as a rule, gives no physical sign during life.

When secondary growths occur in the liver, the organ is enlarged irregularly, and a well-defined tumour may be discovered on the upper surface of the organ in the right or left lobes, or the edge of the liver may show definite nodules. Some of the nodules may be umbilicated, and they are painful and tender. In its connection with cancer of the stomach it may be said that irregular enlargement of the liver in some cases is the only physical sign observed during life—the case is diagnosed as one of cancer of the liver, and subsequently it is discovered that the growth in the liver was secondary to primary disease in the stomach. In such cases, the stomach usually becomes adherent to the liver (see Diagnosis, p. 490.

Cancerous nodules in the peritoneum secondary to cancer of the stomach may be shown during life only by tenderness over the abdomen, varying in extent; but in some cases it is associated with the development of ascites, which may be slight, and usually is so, although it may also be well marked.

Secondary growths in the lung are frequently only small nodules, and are discovered mostly at the post-mortem examination. They may, however, give physical signs, usually at the base of the lung, such as deficient movement, dulness, and alteration of the breath sounds, with crepitations.

Secondary deposits are usually most evident towards the latter end of the disease, and occur most frequently in the liver.

2. *Jaundice* occurs in about 5½ per cent of the cases, but it usually appears towards the latter end of the case, and is due to an affection of the glands near the pylorus or to disease of the liver itself. In some cases the jaundice diminishes towards the end of life, but it does not generally disappear altogether.

3. *Œdema* of the legs occurs in about 12 per cent of cases, and is usually due to the general condition (anæmia) of the patient. It may, however, be due to pressure on the inferior vena cava or to thrombosis of that vein.

4. *Abscesses and other Complications.*—Abscesses are frequently small and only discovered at the post-mortem examination, but a large subphrenic abscess arising from perforation may be formed. The signs of this have already been considered (see Chapter XV. p. 446).

The symptoms of the other complications of carcinoma need not be considered in detail. Pneumonia and bronchitis may occur towards the end, and the physical signs of fatty degeneration of the heart may be shown in dilatation of the left ventricle and in increased frequency or irregularity of the cardiac rhythm. Albumin in the urine may indicate the presence of renal disease, but not infrequently renal disease is only discovered at the post-mortem examination. The presence of cardiac degeneration and renal disease constitute a danger in all cases of malignant disease, which must be borne in mind.

COURSE, DURATION, AND PROGNOSIS.

Course of the Disease.—The course of cancer of the stomach is one of downward progression, as in all cases of malignant disease which are not treated by operation. The symptoms, however, are not continuous; the pain, for example, frequently diminishes towards the end of the disease, vomiting and hæmatemesis vary, and the appetite may be regained to a certain extent. Improvement may be noticed from time to time, an improvement which is chiefly due to the treatment of the functional disturbances of the stomach. Thus by counteracting bacterial fermentation of the food, by careful methods of feeding, and by the use of remedies which increase the digestive power of the organ, patients may improve and gain in weight. The chief symptoms of the disease are still present, but by dieting and the improvement of the digestion more food is assimilated, and the loss of weight is prevented. It is important to remember this point, as it is not infrequently considered that when cancer affects the stomach the loss of weight must be a continuous one.

Duration and Prognosis of the Disease.—No recovery is possible in cancer of the stomach. The disease lasts a varying time; it may kill rapidly within a few months, or death may be delayed twelve months or even as long as thirty-six months from the onset of the symptoms. The average duration is probably about eighteen months. No prediction can be made as regards the time which any individual case will last, the duration depending on factors which cannot be always discovered by clinical means; such, for example, as the rapidity of the growth. The prognosis as regards duration is, however, influenced by the condition of digestion, and by the presence or absence of complications. In some cases the digestion of food, although not good, is possible to a certain extent; whereas in others, when there is stenosis of the cardia, diffuse infiltration of the organ, or great pyloric stenosis and dilatation of the organ, the assimilation of food is so interfered with that the prognosis must be very serious. In any complication such as secondary growths, or the formation of a subphrenic abscess, or the incidence of bronchitis and pneumonia, the immediate prognosis is serious. In those cases which are associated with a dilated and fatty left ventricle, sudden death occurs from cardiac syncope.

Diagnosis.

A suspicion of cancer of an important organ arises when in a patient of middle age, or beyond, there is progressive wasting, weakness, and anæmia without fever, and where there is no sign of wasting disease elsewhere, such as tuberculosis, chronic renal disease, and diabetes. In such a case the combination of symptoms which has been described as the effect of a malignant growth must be borne in mind, as in the absence of any sign of other wasting disease, they are indicative of cancer of an important organ. The difficulty arises in locating the malignant disease, as to whether it is in the stomach or in any other of the abdominal organs. In some cases malignant disease of the stomach is shown by no physical signs in the abdomen, but is associated with indefinite dyspeptic symptoms, there being no great epigastric pain and no hæmatemesis. In such cases, the diagnosis of cancer of the stomach must be a tentative one, but the following points deserve consideration. Cancer of the stomach is probable if the symptoms are out of all proportion to the weakness, anæmia and wasting present, if they have had a definite origin, if loss of appetite is a prominent symptom, and if the examination of the process of digestion by means of a test-meal shows that there is a deficiency in the secretion of hydrochloric acid, and an excessive amount of lactic acid is present.

It cannot be too clearly stated that no diagnosis of a serious case of disease of the stomach is possible without a complete examination of the body, more especially of the thorax, of the abdomen and pelvis, and of the urine. As important as an examination of the stomach are the ordinary methods of examination of these parts. The effect of cancer of the stomach on the process of digestion is usually well marked, and can only be determined by a chemical examination of the vomited matters, or by the use of a test-meal in the manner previously described. There does not appear to be any reason why the contents of the stomach should not be examined as frequently and carefully as the urine. In the diagnosis of Bright's disease the examination of the urine generally gives important results and is essential, but some cases of Bright's disease, for example the granular contracted kidney, may be diagnosed without an examination of the urine, and albuminuria may be absent. Similarly with the cancer of the stomach, some cases can be diagnosed by the symptoms and physical signs in the abdomen and some cannot; and in these latter cases the examination of the process of digestion and the stomach contents is not only of very great value but is essential for the diagnosis.

Diagnosis of Cases with no Tumour but with Dilatation of the Organ.—These cases often present great difficulty in diagnosis, inasmuch as cases of simple and permanent dilatation due to catarrh or old ulcer are associated with the same train of symptoms as cancer of the stomach and dilatation, and may indeed end fatally by exhaustion, the actual condition being discovered only after death. In this case the application of a test-meal is not of great diagnostic value, inasmuch as in simple dilatation there is a diminished digestive activity and frequently bacterial fermentation of the food. In some of these cases, therefore, a diagnosis of cancer is not possible when the patient is first seen or even after some weeks' observation, but in other cases it is possible by a consideration of the following points. In the first place, when the dilatation is due to subacute catarrh of the stomach, although the general symptoms may be very severe and anæmia well marked, there is great excess of mucus in the stomach as shown by the vomit, and mucus may be passed in the motions; there is no hæmatemesis and the patient soon improves under treatment. It may be that in some of these cases suffering from catarrh, a malignant growth is the primary affection, but in the absence of a tumour this cannot be diagnosed.

Secondly, cases of simple dilatation may be distinguished from those that are due to cancer by the fact that they are of longer duration; they may have existed, for example, for two or three years, or severe stomach symptoms may have existed for even longer, as in cases which are due to the contraction of old ulcer or a pre-existing catarrh.

Cases of Cancer with a Tumour.—In cancer of the stomach where there is no tumour present there are serious difficulties in diagnosis, and indeed, except in a few cases, the diagnosis of cancer of the stomach in the absence of a tumour is a matter of conjecture. Even when a tumour is present there may be difficulty in the diagnosis. In the diagnosis of tumours, it has to be considered whether the tumour is in the stomach itself, or whether it is in one of the neighbouring organs, the liver, pancreas, or spleen.

Liver Tumours. — Cases of difficulty mainly arise in those cases where a stomach tumour is adherent to the liver itself. In such cases the stomach tumour is frequently not discoverable by palpation, and only the signs of enlargement of the liver with a localised tumour in the right or left lobe, or nodules along the anterior edge are discovered. The diagnosis of malignant disease of the liver being evident from those general symptoms and physical signs which are characteristic of cancer of the liver, the question arises, Is there a stomach tumour situated at the pylorus?

The following points aid in the diagnosis. There are the symptoms referable to the stomach which have been previously described as present in carcinoma of the organ. The important signs are not the pain, which is present in cancer of the liver as well as in that of the stomach, not the vomiting, which may also be caused by the liver disease, but the occurrence of hæmatemesis and "coffee-ground" vomiting and the results of the examination of the process of digestion. Moreover, the presence of great dilatation of the stomach with malignant disease of the liver is suggestive of cancer of the stomach.

Tumours of the Pancreas.—Tumours of the pancreas are situated in the middle line, and they may be either malignant or cystic. They are not common, and in malignant disease the tumours are small; whereas a cyst may be very large, occupying a large portion of the abdomen. In cystic disease the absence of the symptoms of malignant disease is an important diagnostic sign, and the absence of symptoms directly referable to the stomach is also a point to be noted. Malignant disease of the pancreas most usually occurs in the head of the organ, the tumour being situated to the right of the pylorus. This is not a very important point, however, since in movable tumours of the pylorus the mass may be felt in the position of the pancreas. Inflation of the organ does not necessarily reveal a dilated stomach and the tumour disappears. In this case, however, it is not absolutely certain that the tumour is not in the stomach, because if it is in the posterior wall of the organ it will also disappear on inflation. In some cases of pancreatic disease the stools are clayey even when jaundice is absent, and this may be an aid in the diagnosis.

Disease of the Omentum.—Thickening of the omentum occurs both as the result of tubercular peritonitis and of malignant disease, chiefly scirrhus. The conditions of the stomach from which this thickened omentum has to be distinguished are diffuse carcinoma of the organ and cirrhosis. In both the physical signs are somewhat similar, and consist in an irregular, nodular, and hard mass stretching across the abdomen, ill-defined at the right and left extremities. In the case of the stomach the mass is rather higher than in the case of thickened omentum. This is, however, not a point of great importance. The diagnosis has to be made from a consideration of the following points. In tubercular peritonitis the age of the patient may settle the point, since the disease occurs in young adults; although it may occur at the age at which cancer is frequent. The history of the illness may be diagnostic; tubercular peritonitis running a very chronic course, often practically painless and frequently associated with diarrhœa of greater or less severity, and with fever which may be very irregular in type. Lastly, the presence of tubercular disease elsewhere, *e.g.* in the lungs, is important. The combination of such symptoms indicates the diagnosis. In cancer of the omentum the symptoms of malignant disease are the same as those in diffuse carcinoma of the stomach; the diagnosis is, however, made chiefly from a physical examination. The position of the mass, as has been said, is not of very great importance in the diagnosis, nor are its other characteristics—its irregularity, hardness, and tenderness. These are present in both conditions. But a diagnosis may be made either from the presence of the severe stomach symptoms which have already been described, or from the results of inflation of the organ. The stomach in diffuse cancer cannot be inflated, and inflation may cause great pain, whereas in cancer of the omentum the stomach can be inflated unless it is involved by the disease, and the inflated stomach lies above the tumour and not below it. Dilatation of the organ may be present in disease of the omentum. Examination of the process of digestion will show in these cases of diffuse cancer the great diminution in the secretion and activity of gastric juice.

Tumours of the Spleen.—Tumours of the spleen can only be mistaken for tumours of the stomach when these occur as a diffuse nodular mass over the cardiac pouch. In such cases the diagnosis is made from the following considerations. Great enlargement of the spleen, which is the only condition that can be mistaken for cancer of the cardiac end of the stomach, occurs in adults as a result of malaria and of leucocythæmia. In the case of malaria the history of ague attacks, and frequently the coincident enlargement of the liver, will serve for the diagnosis, as well as the improvement in the condition of the patient as regards the general condition and the size of the splenic tumour from treatment by quinine or arsenic. In leucocythæmia the condition of the blood, that is, the diminution in the amount of hæmoglobin associated with the great increase in the number of white corpuscles serves for the diagnosis. In leucocythæmia it must be remembered that hæmatemesis may occur, but the examination of the blood is sufficient for the diagnosis of the condition. Primary malignant disease of the spleen is unknown.

Tumours of the Stomach itself.—It has been stated that these may be discovered by physical examination either at the pylorus, along the lesser curve, along the cardiac pouch, or in the body of the organ, and they are felt either as nodules or as diffuse masses, nodular in parts and tender. A large tumour of the stomach is always carcinoma.

The diagnosis with regard to pyloric tumours rests between simple fibroid stenosis of the orifice and stenosis due to malignant disease. The condition is one that is not always easy to differentiate. Pyloric stricture may be due to the contraction of an ulcer, either of the simple chronic ulcer or of an ulcer produced by mineral acid poisoning; or it may be due to malignant disease, either to a scirrhus surrounding the orifice or to a fungating mass situated near the pylorus. The diagnosis has to be made from the following considerations:—The presence of the symptoms of malignant disease may at once diagnose malignant stricture from fibroid stricture; but in fibroid stricture there may be great emaciation owing to the starvation of the patient, and anæmia may be a well-marked symptom. The history of the illness may also be distinctive. In simple stricture of the pylorus the history of ulcer may be clear; that is, there was a period of the patient's life during which there was a well-defined localised pain after food and vomiting, with hæmatemesis or melæna; then the cessation of these symptoms, followed by the symptoms and signs peculiar to dilatation of the organ. In cancer of the stomach the history is quite different from this. There is a definite origin, reckoned by months but not by years, and there is no definite remission of the pain and no cessation of the symptoms after the first onset to so great an extent as in fibroid stricture of the pylorus. The duration of the case is of great importance; in fibroid stricture severe stomach symptoms may have lasted for months or years without any great effect on the nutrition; in cancer the duration of the symptoms is reckoned by months alone. The age of the patient may at once decide the point, inasmuch as simple fibroid stenosis of the pylorus occurs usually in young adults, and cancer in middle or old age. It must be remembered that colloid cancer of the stomach also occurs in young adults, but the tendency of colloid cancer is to form a large tumour, that of fibroid stricture to form a small tumour.

Physical Examination.—If the pyloric tumour is large and nodular it is carcinomatous and not simple stricture; but a mistake may arise in those cases in which fibroid stricture is associated with dilatation of the pyloric pouch. In these cases a small thickening may be found near the right hypochondrium, in the position of the pylorus, and behind the first segment of the right rectus there is an ill-defined resistant mass, which gives the sensation of a tumour. In this case inflation of the organ may be of service in the diagnosis, inasmuch as the pyloric pouch will become greatly distended, while if the thickening were due solely to a tumour it would not distend at all, or only to a very slight extent. In both malignant stricture and simple stricture the stomach is dilated, the process of digestion is imperfect, and bacterial fermentation may be present; consequently these points do not aid one in the diagnosis. A small cancer of the pylorus and a fibroid stricture may both be movable; and the diagnosis between the two therefore rests almost solely on the history of the case and on the size of the tumour.

It must be remembered also that simple stricture of the duodenum produces the same results as stricture of the pylorus, and the tumour may be felt in the region of the duodenum, and be undistinguishable from a pyloric tumour, except that it is never movable nor does it move with respiration. Pressure on the duodenum by a malignant growth in the head of the pancreas or in the peritoneum and glands around will also produce the same physical signs as those of malignant stricture of the pylorus. Cancerous peritonitis in the upper part of the abdomen is always secondary to a growth in the abdomen, which is frequently large and easily distinguishable as malignant.

Diagnosis from other Stomach Conditions—(1) *From Ulcer of the Stomach.*—Although there may be cases in which doubt arises as to the diagnosis in these two conditions, yet in the majority of instances the distinction between the two is easy. The age of the patient may be distinctive, cancer occurring in middle age and beyond, ulcer occurring in young adults, although it may occur in middle age, and in women more frequently than in men. The chief points in the diagnosis consist in the history of the illness, in the symptoms produced, and in the effect on nutrition. The history of the illness in carcinoma is frequently of definite onset, and when in ulcer the illness has a definite commencement, it is by way of an attack of hæmatemesis, which is never the mode of commencement in carcinoma. The symptoms in ulcer and carcinoma are widely different. In cancer the pain is continuous, frequently in paroxysms; and although this may be so in rare cases of ulcer, yet the pain in this disease is directly related to the ingestion of food which excites vomiting. It is also associated with localised tenderness in the epigastrium and back, and thus differs from the pain in carcinoma. Ulcer is also characterised by attacks of hæmatemesis of bright blood or dark blood in clots, while in carcinoma the bleeding is slower, and the tendency is to "coffee-ground" vomiting. The effect on nutrition is important; in both anæmia may be present, but progressive wasting is a well-marked symptom in carcinoma.

Lastly, the process of digestion as seen from an examination of the vomited matters or as observed by means of a test-meal is widely different in the two cases. In ulcer there may be an excess of hydrochloric acid, or this may be in small amount or even deficient, but in the majority of instances the chemical process of digestion is good. In cancer there is a tendency to deficient secretion of gastric juice, to deficient motor activity, and to bacterial fermentation, which is shown by the presence of lactic and other organic acids in the stomach contents. In old ulcer where there is dilatation or fibroid stricture of the pylorus, there is greatly deficient functional activity of the stomach, and some difficulty in the diagnosis may arise. The diagnosis has already been considered, and rests on the history of the case, as one showing the presence of an ulcer at a previous period of life, and not so much on the physical diagnosis of the present condition. It must be remembered, however, that carcinoma may be present in a patient who has been the subject of ulcer (p. 460).

2. *From Chronic Catarrh.*—Chronic catarrh existing in middle-aged people gives rise to difficulty in the diagnosis from carcinoma only in those cases where a tumour is absent in the latter disease. In both cases the stomach may be dilated, though not so frequently in chronic catarrh as it is in malignant disease of the stomach. The age of the patient is of no value in the diagnosis, but a long continued illness

with intervals of remission and of exacerbation of the symptoms indicates chronic catarrh, and the contrary indicates carcinoma, while in both there may be wasting and anæmia. The results of treatment are also of great importance. In chronic catarrh the patient derives great benefit from treatment, gaining in weight and the appetite improving; and this improvement may be so marked as at once to distinguish the case from one of carcinoma. The process of digestion in the two conditions may be undistinguishable; there being in both a deficiency of hydrochloric acid and an excess of mucus in the stomach, with a delay of food in the organ. The occurrence of bacterial fermentation is more frequent in cancer than in chronic catarrh.

Subacute catarrh when it occurs in middle age can rarely be mistaken for carcinoma. It may produce a profound effect on the nutrition of the patient and cause wasting and anæmia, and it may have a definite origin; but the following points serve to distinguish the two conditions. In subacute catarrh the pain in the abdomen is in direct relation to the ingestion of food, and there is diffuse tenderness over the stomach region; vomiting is present, and there is rarely any hæmatemesis. The stomach contains a large excess of mucus, and mucus is present in the stools; the process of digestion may be practically absent. It is possible that in some cases subacute catarrh may be added to cancer of the stomach, but in these cases no diagnosis can be made in the absence of a tumour. The great improvement which occurs on the adoption of a proper treatment in subacute catarrh serves as an additional point of distinction between the two conditions.

3. *From Abdominal Aneurysm.*—One condition must be mentioned which sometimes gives great difficulty in the diagnosis, viz. when carcinoma of the pylorus forms a large tumour and is pulsatile. In this case the diagnosis has to be made from aneurysm of the abdominal aorta or of one of its branches. Physical examination is of no great aid, for the pulsation in both cases may be undistinguishable, and the absence of expansile pulsation does not exclude the presence of abdominal aneurysm. The diagnosis has to be made from the following considerations. The history of the patient may

be distinctive: it is the history, in cancer of the stomach, of a wasting disease producing great anæmia and weakness and associated with stomach symptoms, such as pain, vomiting, and hæmatemesis. In abdominal aneurysm there is a chronic illness, the chief and frequently the sole feature of which is pain. The pain may be undistinguishable from that of carcinoma, and vomiting may be excited by the pressure of the aneurysm on the stomach, but hæmatemesis is usually absent. The definite origin of the stomach symptoms and their continuation is in favour of cancer of the stomach and not of aneurysm, since the stomach symptoms resulting from an abdominal aneurysm situated near the pylorus are only casual and intermittent. Inflation of the organ may be of service in showing that the tumour in the case of aneurysm is not situated at the pylorus, but behind it and behind the stomach. Examination of the process of digestion may decide the diagnosis by showing the great deficiency which exists in cancer of the stomach.

Treatment.—The treatment of cancer of the stomach is only palliative. None the less it is of great importance, inasmuch as by treatment the functional deficiency in digestion may be to some extent remedied, and the patient enabled to assimilate sufficient food for him to lead a moderately comfortable existence. The treatment is both medicinal and dietetic, but chiefly dietetic.

Medicinal treatment is directed to the relief of pain by means of sedatives, chiefly by the hypodermic injection of morphine (p. 301), to the relief of constipation (p. 309), and of bacterial fermentation of the food (p. 305). In many cases, however, chiefly in the earlier stage, the administration of acids and alkalies is of benefit (p. 290), and bitter tonics such as quinine, nux vomica, quassia, and condurango bark are of value in improving the appetite. The greatest benefit, however, arises from the counteraction of bacterial fermentation by the daily washing out of the stomach (p. 359), and by antiseptic remedies, as by this means the stomach is enabled to digest some food which would otherwise be lost to the organism.

Dietetic treatment is of great value, and is to be prescribed on the lines which have been previously discussed (Chapter XII. p. 351), either by the adoption of rectal feeding for a time, or by the administration of peptonised foods by the mouth, or of easily digested food articles, such as small quantities of milk, scraped steak, or pounded fish.

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